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SPASTICITY AND THE FRONTAL LOBES: A REVIEW 1017
John F. Fulton

PRESENTATION OF THE HENRY JACOB BIGELOW MEDAL 1024

ADDRESS FOLLOWING THE PRESENTATION OF THE HENRY JACOB BIGELOW MEDAL 1026
Edward W. Archibald

PARA-AMINOBENZENESULFONAMIDE AND ITS DERIVATIVES IN THE TREATMENT OF BETA HEMOLYTIC STREPTOCOCCUS INFECTIONS OF THE MIDDLE EAR AND MASTOID 1033
Carlyle G. Flake and *Benjamin W. Carey, Jr.*

SURGICAL TRAINING IN THE NON-UNIVERSITY HOSPITAL 1039
Lucius C. Kingman

IODINE RESPONSE AND SOME OTHER FACTORS IN THEIR RELATION TO MORTALITY IN THYROTOXICOSIS 1041
J. Lerman

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL 1045

EDITORIALS
Christmas 1937 1052
Legal Medicine 1052

DEPARTMENTS
Massachusetts Medical Society 1053
Miscellany 1055
Correspondence 1058
Recent Death 1059
Report of Meeting 1059
Notices 1060
Book Reviews 1061

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SPASTICITY AND THE FRONTAL LOBES

A Review

JOHN F. FULTON, M.D.*

NEW HAVEN, CONNECTICUT

INTRODUCTION

FROM the time of Hughlings Jackson,[13] but more particularly since the publication in 1898 of Sherrington's[27] paper on decerebrate rigidity, the spasticities and rigidities observed in human beings have been looked upon as "release phenomena." The brain-stem and medullary levels normally under the domination of the higher centers are allowed to function unimpeded when certain parts of the forebrain or its connections are destroyed. Spasticity itself, that curious dough-like resistance which occurs in certain states of release, especially in the antigravity muscles, is associated with increased stretch and tendon reflexes, clonus and the "clasp-knife" phenomenon; occasionally the neck reflexes of Magnus[17] can be demonstrated. It has been pointed out by Walshe[30] that these features of the spastic state are seen also in decerebrate rigidity of cats and dogs, and he concludes accordingly that spasticity and decerebrate rigidity must result from operation of similar mechanisms.

My own interest in the problem began about 1926 as a result of my contact with Cushing's clinic at the Peter Bent Brigham Hospital, where it was taught that superficial cerebral lesions often produced paresis without spasticity, while deep lesions, particularly those of the capsule, more generally gave rise to spastic hemiplegias. Experimental studies of the problem in cats and dogs[15, 34] had proved disappointing, since the entire cerebral hemisphere of such animals can be removed without causing spasticity. It is undoubtedly significant, however, that *limited* destruction of the anterior frontal region of dogs and cats gives rise to moderate spastic resistance in the extremities opposite to the lesion,[34] but the *quality* of the resistance does not closely resemble that observed in spastic human beings. With monkeys and chimpanzees, however, the situation is quite different, since it is possible to reproduce in these animals syndromes of release which simulate in almost every detail those encountered in human subjects.

If spasticity is a release phenomenon, one wishes to know something of the origin of the anatomic projections normally suppressing the centers involved. Motor projections arise from many parts of the cerebral mantle; these projections in all the higher animals may be divided, as Foerster[4, 5, 6] points out, into two principal groups: (1) the corticospinal or pyramidal projections which form the corticospinal tracts, and (2) the extrapyramidal projections passing to various subcortical and brain-stem centers. In man and monkey the principal areas of motor projection lie in the frontal lobes; that is, in the part of the hemispheres lying ahead of the central sulcus (Fig. 1). Some motor projections arise also from the parietal and temporal lobes[21, 24, 16], but their functions are almost entirely unknown.

The Pyramidal Projections

The cells of origin of the corticospinal system arise in the frontal lobes from the cytoarchitectural field designated in Brodmann's map as Area 4. In man this occupies the entire anterior wall of the central sulcus, emerging on the surface of the precentral convolution, most extensively in its superior part (Fig. 1). Area 4, by virtue of its corticospinal connections, includes the principal part of the classical motor area. The cells of origin of the corticospinal tract, first described by the Russian neurologist Vladimir Betz, occupy the fifth cell layer of cortical gray matter, and the dendrites of these enormous cells branch into all six layers so that, like funnels, they collect impulses from a relatively large region. Area 4 is defined as that part of the precentral convolution containing Betz cells. It is generally believed that no corticospinal fibers arise from other cells. Holmes and May,[12] who were largely responsible for this doctrine,

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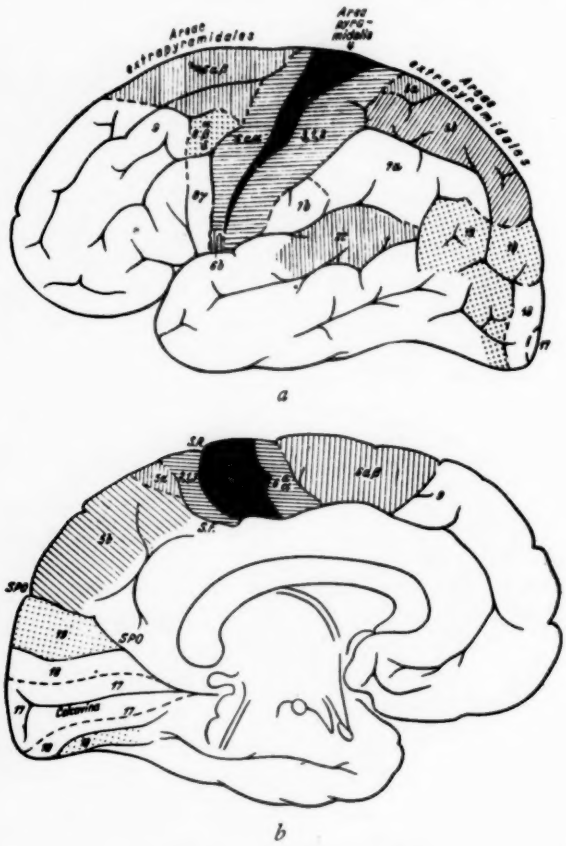
pointed out that when the pyramidal tract was cut through in the spinal cord, the Betz cells appear to be the only ones which undergo chromatolytic change. It is difficult, however, both in monkeys and in chimpanzees, to determine the exact anterior margin of Area 4 because the more rostrally situated Betz cells are smaller than those in the central sulcus, and their population in a given section diminishes at the anterior extent of the



a. Lateral surface of hemispheres.
b. Medial surface of hemispheres.

Fig. 1. *Foerster's[6] diagram of the principal cytoarchitectural areas of the human brain, based on Brodmann's numerical designations as modified by the Vogts. Areas 4 and 6 of the frontal lobes are histologically similar except for the absence of Betz cells in Area 6. Area 4, which gives origin to the pyramidal tracts, is shown in black; it extends to the bottom of the central sulcus. Area 6 is divided into an anterior part, Area 6 aβ, and a posterior strip, Area 6aα.*

field. In monkeys Betz cells may occasionally be found as far forward as the anterior margin of the premotor region (Area 6, Fig. 1), but the principal region of difficulty lies along a narrow band of transitional precentral cortex shown in Figure 2, which has been designated the "strip region" (Hines[11]). The functional significance of this region will be described below.

Area 4 itself has been subdivided into Areas 4*a*, 4*b* and 4*c*, denoting respectively the representation of the lower extremity, the upper extremity and the head.

The Extrapyramidal Projection Areas

Omitting for the time being those extrapyramidal projections arising posterior to the central sulcus, the functions of which have not yet been analyzed, attention will be directed to those in the frontal lobe.

Area 4. It is sometimes forgotten that Area 4, in addition to giving rise to the corticospinal projections, has a rich extrapyramidal projection, as

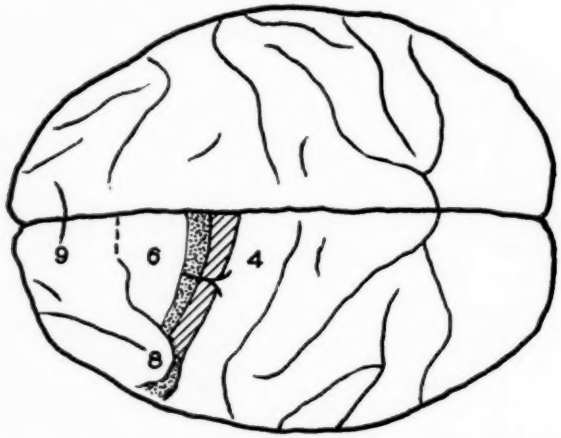


Fig. 2. *A diagram, adapted from Hines,[11] of the macaque cortex showing the relative position of Areas 4 and 6 and the intermediate cortex or "strip region" which lies between them. In most animals the cross-hatched area contains Betz cells throughout most of its extent, and thus corresponds with the anterior part of Area 4; the stippled area may have occasional Betz cells, and in most animals represents the posterior part of Area 6. The strip extends down the medial surface of the hemisphere to the fissure callosomarginalis.*

originally pointed out by Mellus;[21, 22] and according to the recent studies of Paul Levin,[16] these projections are practically identical in distribution and destination with those arising from the premotor region known as Area 6 (Fig. 1).

Area 6. Apart from the aberrant Betz cells which are often found in Area 6 of the monkey, it is probable that this region does not give rise consistently to corticospinal fibers. Levin finds that it contributes heavy projections to the substantia nigra, red nucleus, pontine nuclei, reticular substance, bulb and also some to the hypothalamic area. As already mentioned, these extrapyramidal pathways cannot be distinguished from those arising from Area 4; indeed they appear to be part of a single extrapyramidal projection. There are two distinct corticopontine tracts, one arising from Areas 4 and 6 and passing through the middle

segment of the peduncle, the other of uncertain origin in the frontal lobe (probably frontal pole, Levin[16]), which passes through the inner segment of the cerebral peduncles.

Area 8. From Area 8 a compact bundle takes origin which passes through the capsule at the genu of the globus pallidus and into the medial part of the peduncle, terminating in the substantia nigra and tegmentum, evidently passing to the eye muscle nuclei.[10]

Areas 9, 10, 11 and 12. The principal connections of this region are intracortical. However, from Area 9 there is a fine-fibered projection which passes through the anterior limb of the internal capsule to the medial nucleus of the thalamus; it also sends fibers to the caudate nucleus and pons, the principal connection being corticothalamic.[16, 23]

REGIONAL ABLATION AND SPASTICITY

Until recently it has been tacitly assumed, especially among clinical writers, that spastic manifestations result primarily from lesions of the corticospinal projections. In clinical neurology it has long been a working hypothesis that an upper motor-neuron lesion brings spasticity in addition to paralysis, and that a lower motor-neuron lesion is flaccid. Although exceptions were encountered, from time to time, the nature of the pathologic process was so often one giving a complicated and ill-defined anatomic lesion that its explanation could be determined only by physiologic experiments on anthropoids. In attempting to carry out a systematic study involving the regional ablation of the principal cytoarchitectural areas of the frontal lobe, I was surprised, in 1929, to find in the monkey that lesions restricted to Area 4 caused paresis of volitional movement without obvious spasticity. This observation has been the subject of considerable discussion,[31, 32, 33] more than I had expected it to evoke. Several years have passed since the observation was made, and it now seems worth while to review the present status of the problem.

Area 4 and the Pyramidal Projections

If Area 4a, the leg representation, be removed from an adult macaque monkey, all observers are agreed that the extremity passes at once into a state of profound flaccid paralysis.[2, 9, 11] The knee jerks immediately become depressed and superficial reflexes are difficult to elicit, but after several days the reflexes become more active and motor power begins to return, first at the hip, later at the knee, and much later still in the digits. After the return of motor power, postural resistance may become virtually normal, although on passive manipulation at the hip and knee one can generally demonstrate a diminished passive resistance several months after such a lesion. When the corresponding experiment was carried out on a chimpanzee, the degree and duration of the flaccid paresis were even greater than in the monkey; indeed, the lower extremity of one chimpanzee exhibited diminished postural resistance at the hip and knee for five or six months, but it then became indistinguishable from normal. On the basis of these observations, which were begun in 1929 and first published in 1934, Kennard and I[9] suggested that, contrary to clinical opinion, lesions primarily involving the pyramidal tract gave rise to flaccid pareses. Our position was considerably strengthened by the fact that when we subsequently removed Area 6 from a previously flaccid Area-4 preparation, the affected extremity became immediately spastic, showing increased tendon reflexes, the clasp-knife phenomenon, clonus and other reflex changes described below. The changes, moreover, proved to be enduring.

The controversy which developed after these observations were published has had the virtue of bringing out a number of new and significant facts. In the first place, I was myself somewhat disturbed, not so much by the criticisms of Walshe,[31, 33] since these lacked the support of experimental study, but by the fact that we had at about this time observed the effects of lesions involving Area 4*b*, the arm representation, instead of 4*a*. In a chimpanzee following such a lesion the arm became flaccid at first, just as in the case of the leg, but later the extremity developed mild spastic resistance. Such a state of spastic resistance could always be immediately augmented, however, by extending the lesion anteriorly into Area 6. Insofar as Area 6 augmentation spasticity was concerned there was no doubt about the validity of our position, but the arm had appeared to behave somewhat differently from the leg in response to ablation of Area 4.

In monkeys, moreover, we had missed a significant phase of recovery of motor power in the arm, as well as in the leg, following lesions of Area 4. Denny-Brown of London, who visited my laboratory last summer (1936), studied afresh the phases of recovery of motor power in monkeys following lesions of various parts of Area 4. With Botterell,[2] of Toronto, he made lesions of Area 4, some subtotal and others complete, in macaques, and the events accompanying recovery of motor power were observed daily for periods varying from two to four months.

In one animal, an adult macaque, Areas 4*a* and 4*b* (arm and leg) were removed well up to the posterior margin of Area 6. For twenty-four hours there was virtually complete paralysis of the right upper and lower limb; motor power then began to appear at the hip and shoulder, but the limbs offered no resistance whatsoever to passive

manipulation. After a week, when motor power had returned at the hip, knee, shoulder and elbow, the lower extremity began to assume a slightly extended posture and a somewhat flexed attitude, but resistance to passive movement was still meager at all joints, including the wrists and digits. After two weeks the deep reflexes were still somewhat sluggish, and the resistance to passive movement of the proximal joints continued to be below normal. At this time, however, a finger jerk could be obtained on the right side. Two days later there was some resistance in the ankle and toes, and the fingers on the right side were beginning to become clawed; the knee and ankle jerks on the right also became more active. By the end of the third week the fingers and toes were strongly clawed and the ankles were spastic with clasp-knife relaxation, but there was no appreciable spasticity in the knee or elbow. By the thirty-first day the animal had regained some use of its fingers, and by the thirty-third day there was definite finger movement. With the return of movement in the digits the spastic resistance in the digits became less. The animal was observed for a period of fifty-eight days, after which the second motor area was removed; the animal was sacrificed seventeen days later. The secondary procedure was followed by a similar but more intense paresis, which was flaccid in the proximal joints, just as on the other side.

In another similar animal with a more posterior lesion of Area 4, subsequent removal of the anterior strip of Area 4 and the posterior millimeter or so of Area 6 caused the appearance of moderate spasticity at the elbow and shoulder.

Denny-Brown and Botterell thus established the occurrence of a transient phase of distal (digital) spasticity following lesions restricted to Area 4, but so far as proximal joints are concerned, the paresis is flaccid in character, as Kennard and Fulton had originally observed, provided the strip region is not wholly removed.

Quite independently of Denny-Brown and Botterell, Marion Hines[11] announced in April, 1936, that generalized spastic resistance followed in the macaque if a strip of cortex including the anterior 3 millimeters of Area 4 was removed from that part of the cortex in which the Betz-cell population is relatively sparse. Her criteria of spasticity were essentially those adopted by Walshe,[30] that is, increased tendon reflexes, increased passive resistance, clasp-knife phenomenon, clonus, and so on. The spastic resistance was generalized throughout the entire extremity, being particularly conspicuous in the shoulder and elbow, and the postural disturbance was said to be enduring.

From the observations of Denny-Brown and Botterell and of Hines it can thus be concluded that the posterior four fifths of Area 4, if removed in a macaque, induces a flaccid paresis in the proximal joints, but this is characterized, from the second through the fourth week after operation, by a phase of digital spasticity. When a strip lesion is made in the anterior portion of Area 4, a mild spasticity occurs in the proximal joints; and finally when a total lesion of Area 4 is made in the macaque, spasticity of a mild degree may develop in all joints; but how long it endures remains to be determined.

Thus far in the discussion I have carefully avoided any implication as to whether the transient spasticity seen with Area-4 lesions is due to an interruption of the pyramidal or to destruction of the extrapyramidal projections.

Area 6 and the Extrapyramidal Projections

A lesion sharply restricted to Area 6 either in the macaque or in the chimpanzee is followed in our experience[7, 9] and in that of Denny-Brown and Botterell,[2] by a transient weakness in the affected limb. In the macaque this weakness is indeed very slight so long as one does not encroach upon the strip region.[2] The extremities are held in positions of slight flexion of the elbow and generally of some little extension of the knee in the macaque, and there is flexion of the knee in the chimpanzee. Tendon jerks, however, are considerably increased in the chimpanzee. On passive movement of the knee and elbow, as well as of the shoulder and hip, a soft plastic resistance is encountered in both the flexors and the extensors. This continues in the monkey for a week or sometimes less, but if the lesion is bilateral it may endure for several months. In the chimpanzee all intervals are longer than in the macaque. Denny-Brown has pointed out that this resistance is not akin to spasticity as such, but is similar to a true extrapyramidal rigidity; that is, it is plastic and equal in quantity through all phases of the passive movement from beginning to end, and tends to be evenly distributed in flexors and extensors. In the macaque the deep reflexes are not markedly increased, but this has not been the case in the chimpanzee.

When an Area-6 lesion of the type just described is superimposed upon a complete lesion of Area 4, especially in chimpanzees, a conspicuous degree of rigidity results, affecting all the muscles and all the joints.[7, 9] It appears therefore that we are dealing, when lesions of this sort are carried out seriatim in the frontal lobes, with a pari passu release of subcortical centers. It is now essential to inquire what part the pyramidal tracts themselves may play in this release from higher control. Is the digital spasticity, for example, due to the interruption of the pyramids themselves or to the concomitant interruption of the extrapyramidal projections?

UNCOMPLICATED SECTION OF THE PYRAMIDS

Various attempts have been made to sever the pyramidal tracts along the medulla oblongata at the level of the trapezoid body, but generally with indifferent success.[20] Recently, however, Mar-

shall,[18, 19] in the Department of Anatomy at Yale, and Tower,[28] of Johns Hopkins, have succeeded in making lesions, in some instances discrete and probably complete, of one or both pyramids by a retropharyngeal approach to the medulla. Working on cats, Marshall reports in addition to disturbances of movement a gross impairment of placing and hopping reactions; he also finds (in certain positions of the body in space) an increased resistance to passive *flexion* of the limbs, and diminished resistance to passive extension. In a supine position, however, the postural resistance of the affected extremities is definitely less than normal in both the flexors and the extensors. In no stage of recovery was a phase of extensor spasticity observed proximally or distally.

Tower's[28] independent findings in the cat confirm in most details those of Marshall;[19] furthermore both investigators have found that, when the homologue of Area 4 is removed from an animal whose pyramid had been previously severed, the affected extremities suffer an added increment of motor paralysis, and show considerable *increase of extensor resistance*. They state that, after pyramid section in the cat, spasticity and other signs of release are absent. Stimulation of the motor cortex, immediately or months after such a section, demonstrates the preservation within that cortex of inhibitory projections, effective on tonic or clonic states present in the limbs, that is, such as those that are induced during ether anesthesia. "Repetition of this lesion in rhesus monkeys produces a similar though graver syndrome of deficit, again without spasticity" (Tower[28]). These investigators found no evidence of spasticity at any stage following pyramid section in the monkey, though evidence of digital spasticity was specifically sought. Last year Botterell succeeded in cutting the pyramid in the monkey by the same retropharyngeal approach; in one animal which was studied for a period of sixteen days and then sacrificed for Marchi studies, the paresis was entirely flaccid both proximally and distally during the postoperative interval.

In summary, then, it is clear from the evidence now available that the extrapyramidal rather than the pyramidal projections from the cortex are those which, when interrupted, give rise to the spastic state. Until there is some positive evidence to the contrary, therefore, I am inclined to look upon the transient digital spasticity of Denny-Brown and Botterell following an Area-4 lesion as a manifestation of the extrapyramidal injury which inevitably occurs when this area is removed.

REFLEX CHANGES FOLLOWING LESIONS OF THE FRONTAL LOBE

From the point of view of the localization of cerebral lesions, it may be useful to summarize what has been observed concerning reflex disturbances which follow regional ablation of discrete areas within the frontal lobe.

Area 4 (Motor Area)

Apart from a generalized depression of reflexes, the only enduring reflex change of significance following ablation of Area 4 in a chimpanzee is a Babinski response.[7, 8] During the first days following such an ablation, plantar stimulation causes a slow extension of the hallux, accompanied by slow extension of the other four toes, and a generally concomitant reflex contraction of the hip and knee flexors, giving a withdrawal response. When the lateral surface of the foot is stimulated, a similar reaction occurs which in clinical neurology is usually referred to as a Chaddock reflex. This, it is generally believed, is merely an extension of the reflexogenous zone of the Babinski response. In man and monkey, and in a chimpanzee which I studied with Denny-Brown (unpublished), the phase of digital spasticity is also to be expected in the fingers; according to Walshe,[32] it occurs in the toes of man following Area-4 lesions, although there was no direct proof in Walshe's case that the lesion was in fact restricted to this area. This is associated with considerable increase in the deep reflexes of the digits, especially of the flexors; the exaggeration at times may be of sufficient intensity to give rise to a Hoffmann response,[3] or to a considerable exaggeration of the Rossolimo sign in the toes. Even at the height of this increase of digital reflex, the arm and shoulder remain flaccid. For reasons indicated above it is probable that the digital changes are due not to the interruption of the pyramid but to a concomitant disturbance of the extrapyramidal projections.

The Strip Region (Transitional Cortex)[11]

The reflex disturbances following removal of the strip have not yet been sufficiently analyzed to allow a categorical statement concerning the reflex changes, since Hines's detailed paper on the subject is not yet available. However, from her recent lecture (Fig. 2), and from the experience of Denny-Brown and Botterell, it is clear that there is a generalized increase in the deep reflexes of the proximal joints and some increase in the distal. Forced grasping does not occur, and judging from unpublished observations on chimpanzees it is improbable that

there is a Babinski response, though a Rossolimo can be obtained.

Area 6 (Premotor Area)

The most conspicuous reflex change following a primary lesion of Area 6 is the appearance of forced grasping, as was first pointed out by Richter and Hines[25] for the macaque. At present I have under observation in the laboratory an adult chimpanzee from which Area 6 was removed in its entirety a few millimeters anterior to Area 4, so that the greater part of the strip region still remains intact. This animal manifested strong forced grasping in the arm and leg for a period of five weeks, a great increase in digital reflexes, a positive Hoffmann reflex of the fingers, and a conspicuous Rossolimo's sign in the toes. There were also vasomotor disturbances of both contralateral extremities. These reflex changes waned after seven weeks and the grasp had almost entirely disappeared after six weeks, but the sign of Hoffmann continued for several months; the Rossolimo sign diminished but never disappeared, while the vasomotor disturbance entirely disappeared. In this and in many other corresponding experiments in chimpanzees, the pyramidal tracts have not been encroached upon, and yet one sees a syndrome striking in character which, except for forced grasping, had in the past been attributed categorically to interruption of the pyramidal tract itself.

The reflex changes just described became enduring when Area 6, the strip region and Area 4 were all destroyed, especially in chimpanzees. One such animal was observed over a period of three years without essential diminution of its symptoms, except for forced grasping; this symptom, however, was still present when the animal became excited.

Other Areas

Although Area 9 sends an extensive projection to the medial nucleus of the thalamus and also to the pons, its ablation with Areas 10, 11 and 12 in both monkeys and chimpanzees is without effect upon reflexes and postural movement unless the lesions are bilateral, in which case they cause a state of motor restlessness (Richter,[25, 26] and others), mild catalepsy and some increase in the reflexes of the trunk (Messimy and Finan, unpublished). Bilateral ablation of these areas also induces a disturbance of quite another category which in the last few years has been studied in some detail by Jacobsen and his collaborators, but the discussion of the frontal association areas from this point of view is beyond the scope of this paper. Observations in chimpanzees indicate that when Area 9 is destroyed after removal of Areas 4 and 6, the hemiplegic rigidity which had resulted from these lesions becomes further augmented.

Ablation of Area 8 (the eye fields) is without effect upon reflexes of the extremities, but such a lesion causes conjugate lateral deviation of the eyes toward the side of the ablation, and the animal tends to rotate in ordinary locomotor movements toward that side.

DISCUSSION

In the light of the work of Hines and Tower, and of Denny-Brown and Botterell, it now becomes possible to harmonize many apparent discrepancies in the recent literature concerning the frontal lobes. In the first place the term "spasticity" has often been used indiscriminately to describe many forms of augmented muscular resistance in man and experimental animals.[14] Denny-Brown has emphasized that in monkeys as well as in men conspicuous differences exist in the *quality* and distribution of muscular resistance which develops after various regional ablations of the cortex. A lesion restricted to Area 6 causes a soft plastic rigidity entirely akin to, but less intense than, certain rigidities of basal ganglia disease. Stretch reflexes are little augmented, and the distribution of plastic resistance is wide; that is, it affects proximal and distal joints alike, and both the flexor and extensor muscle groups are involved.

Lesions more posteriorly situated involving the posterior part of Area 6 and the anterior part of Area 4 primarily affect the antigravity muscles, giving augmented stretch reflexes, with brisk tendon jerks and other features characteristic of the spastic state. When a pure Area-6 lesion is made after such a lesion, an element of plastic rigidity is added to the spastic state, which results in a more intense muscular resistance in the affected limbs. The addition of the element of rigidity, however, may actually diminish the excursion of the tendon reflexes (occlusion), even though the state of resistance of the muscle as a whole is considerably augmented. Unpublished observations on chimpanzees indicate that lesions of the frontal association areas, that is, Area 9, and so forth, if made after Areas 4 and 6 have been destroyed, further increase the rigidity of the hemiplegic extremity.

Since primary section of the pyramids is not followed by any of the above symptoms of release at any period following their section (Tower and Hines[29]), we are led inevitably to the conclusion that spasticity and the plastic forms of rigidity are all phases of extrapyramidal release from cortical control, that spasticity itself represents a regional release from a particular group of cortical projections, and that the extreme forms of spastic

rigidity which appear with hemiplegia in man are undoubtedly due to the combined release from many extrapyramidal projections, especially those of the frontal lobes.

When both frontal lobes are entirely destroyed the thalamic reflex status (Bieber and Fulton[1]), in which all extremities show spastic rigidity is induced, but the *distribution* of the resistance in a given extremity depends upon the animal's position in space, and analysis indicates that the pattern of rigidity depends upon the interaction of body righting, tonic neck and labyrinthine reflex mechanisms. Not until all the extrapyramidal projections from the cortex are destroyed, thus giving complete release of subcortical centers of posture, is the operation of these centers manifest. Occasionally with incomplete release, that is, in capsular hemiplegia, the presence of neck reflexes can be demonstrated. In these circumstances the actual distribution of spastic rigidity depends upon the position of the body in space in relation to the neck.

SUMMARY

1. Spasticity is a state of release from cortical control of subcortical centers of the brain; it is characterized by increased resistance to passive manipulation, especially in the antigravity muscles (extensors of the lower limb and flexors of the upper), and is associated with increased tendon and stretch reflexes, clonus, the clasp-knife phenomenon, and so forth. It is thus to be distinguished from basal ganglia rigidity in which the *quality* of resistance is different and in which these specific reflex changes are not conspicuous.

2. In monkeys and the higher apes the motor projections whose interruption causes spasticity arise in the frontal lobes. Regional ablation of various parts of the frontal lobes in macaques indicates that spasticity occurs in its purest form after removal of a strip of transitional cortex lying between the motor and the premotor areas (Hines). When Areas 4 and 6 are removed in addition to the strip region, an enduring element of rigidity is introduced, causing the tendon reflexes to be masked, and the resistance to passive movement to be greatly augmented both in the proximal and the distal joints; in these circumstances the resistance is no longer restricted to antigravity muscles.

3. Isolated section of the pyramidal tracts in the medulla of a macaque causes a simple flaccid paresis without trace of spasticity in any joint at any time following the lesion (Tower).

4. Ablation of Area 4 posterior to the strip region causes a similar flaccid paresis at proximal joints associated with a transient phase of digital spasticity (Denny-Brown and Botterell).

5. Ablation of Area 6 anterior to the strip region causes forced grasping and a state of soft plastic rigidity equal in quantity from beginning to end of passive movement, which tends to affect flexors and extensors to an approximately equal extent. This resistance has been inaccurately referred to in the past as "spasticity."

CONCLUSIONS

1. Spasticity is unrelated to disturbances of the pyramidal pathways.

2. Spasticity is a manifestation of release from extrapyramidal cortical projections.

3. Pure spasticity in antigravity muscles results from interruption of the extrapyramidal projections situated in the posterior part of Area 6 and in Area 4.

4. When all the extrapyramidal projections of the frontal lobe are destroyed, a much more profound and enduring rigidity occurs which tends to mask spasticity; in these circumstances muscular rigidity becomes generalized, but depends for its precise local distribution upon the position of the body in space (operation of neck, labyrinthine and body-righting mechanisms).

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BOSTON SURGICAL SOCIETY

PRESENTATION OF THE HENRY JACOB BIGELOW MEDAL

A MEETING of the Boston Surgical Society and invited guests in John Ware Hall, Boston Medical Library, on May 21, 1937, was called to order by the president of the society, Dr. William C. Quinby, who spoke as follows:

It is my pleasant duty tonight to give expression to the feelings of the medical profession, as reflected through the Boston Surgical Society, of admiration and affection for our Canadian cousins and particularly for Dr. Edward W. Archibald, of Montreal. Dr. Archibald needs no introduction to this audience. As surgeon to the Royal Victoria Hospital, of Montreal, and as professor of surgery of McGill University Faculty of Medicine, his abilities as a beloved teacher and a productive worker have long been known in all surgical circles in the United States as well as in the Dominion. Furthermore, it has been noted of late as concerns biography that the behest of the chronicler, "Let us praise famous men and the fathers that begat us," has been changed to read, "Let us praise the fathers that begat us, and if we have sufficient literary skill, we may make them famous." I have no literary skill, and certainly need not attempt a biography of Dr. Archibald, famous though he be.

In place of this I had the thought that a brief recital of some few of the notable milestones in the founding of McGill and in earlier medicine in Montreal might serve to give a background to this occasion; for though in the final analysis it is the man, or more often the generation of men, that makes an institution what it is today, we know also that the personality of the institution in turn has its effect on the man, especially if the institution be the medical school of a famous university.

And now, though it is a digression, I address myself to any of you who may be librarians, or who in any way have to do with the arranging of books on shelves; and file a protest against a custom which I feel should be immediately changed. My protest becomes evident through the following experience. In the Widener Library a few days ago I visited the stacks to see what of value might have been written concerning the earlier history of McGill — something relating to the days before the Golden Age of medicine in the years of Osler. Collected under the large heading of "Education" are to be found such writings relating to universities, but I could not locate McGill. I thought that the fault might lie in the "Mc," which is sometimes given a position as though spelled "Mac." But this yielded nothing. I found "Marietta" and "Maryland," but no "McGill." It is always a challenge to see if one can puzzle out the listing habits of libraries, so I suppose that I wasted much more time than was necessary; but I had to give up. Imagine my feelings on again reaching the central desk with my story of frustration: "McGill? Why, that's a *foreign* university! Look under that group." I feel that without question the genus librarian is the last remaining person of presumed sanity on this continent who persists in thinking of McGill, or for that matter of any Canadian university, as "foreign." They are sister universities of whom we are all justly proud; all members of the same family. Foreign, indeed! I know that you agree that my protest is well taken.

James McGill, a prominent citizen of Montreal at the end of the eighteenth century, bequeathed by will in 1811 ten thousand pounds and a considerable parcel of land in trust to the then infant Royal Institution for the Advancement of Learning, for the founding of a university, at least one of the colleges of which should bear the name McGill. A further stipulation withdrew the gift if for any reason the desires of the testator were not carried out within ten years after its receipt. By this generous act a practical step was made toward the erection of the first Canadian university.

McGill was born in Glasgow in 1744, whence, as a young man and shortly before the American Revolution, he went to Montreal, then a town of

about ten thousand people. He prospered as a merchant and as a member of the Northwest Fur Company, and at the turn of the century he was one of the leading citizens of the town, which by then had more than doubled its size, owing in large part to the influx of loyalists from our eastern seaboard at the time of the Revolution. But the population of the town, and in fact of the whole Province of Quebec, was essentially French, though the conquest by the English had become final in 1764. French institutions were strong and wealthy under the control of the older Roman Catholic religious orders continuing under the British policy of noninterference. The Government's attitude toward the minority of English Protestants was paternal but timid and weak, so that educational facilities for those not of the Catholic faith were few and almost without financial support. McGill was one of a group of far-seeing educated men who felt the great necessity of providing for the education of the English population. The result of their efforts appeared first in 1801 in the act which created the Royal Institution for the Advancement of Learning, and in the hands of this body, McGill subsequently left his legacy. He died in 1813; and even as he had foretold, matters concerning the proposed university dragged on, partly through lack of agreement of the officers of the institution, but mostly because of the entire lack of funds. Incidentally it should be noted as an index of the feeling which then existed between the French and English that the will of McGill was contested in the courts by his stepson, the offspring of Mrs. McGill by an earlier husband, one Désrivières. Finally in 1821 under a modified board of trustees the Royal Institution was granted a charter for the foundation of McGill College.

While these educational matters were going forward, medical affairs in the city as concerned the care of the Protestant immigrant sick left much to be desired, so that again through private generosity, after a small beginning in 1818, the Montreal General Hospital was founded in 1821 with an attending medical staff of Drs. William Caldwell, William Robertson, Andrew Holmes and John Stephenson. The latter two, Holmes and Stephenson, were graduates of Edinburgh, while Caldwell and Robertson were British military surgeons. Matters went quickly, so great was the general need of adequate medical care and instruction, and in 1824 there was authorized the so-called Montreal Medical Institution with permission to conduct lectures; in 1829, at a joint meeting of the governors of McGill College (which thus far had existed mostly on paper) with the four physicians above mentioned, the Montreal Medical Institution was engrafted upon McGill University as its medical faculty. In 1832 permission from the Crown was received by this faculty to hold professorships and to confer degrees.

And so the university was launched; but the next thirty years were passed in great poverty and with considerable dissension over one thing and another, until in 1855, under a new charter, Sir William Dawson was appointed principal. To this remarkable man, who led the fortunes of the university for the next thirty-eight years, its present educational pre-eminence is due. As Maude Abbott puts it, "He found a wilderness with one small oasis; he left a vigorous university with seventy-four members on her teaching staff and a thousand students."

Of this group of teachers, none take higher rank or are more deserving of praise than the medical faculty, who are the descendants of that hard-working, earnest quartet of physicians whose so-called "Medical Institution" was engrafted onto the early college in order to give it tangible existence. Brilliant men have left the portals of McGill throughout all the succeeding years to become illustrious as jurists, teachers or scientists; but the doctor, in numbers and prominence, has continuously outshone them all. He it is who has notably "held high the torch" first lighted by the act of James McGill.

Today McGill University impresses one strongly as being a happy university. No doubt the intermingling of the light-hearted French and their "habitant" folklore, so charmingly related in the poems of Dr. Drummond, has done much to relieve the innate dourness of the Scot and the Englishman. Be this as it may, in her extensive grounds enclosing many noble buildings, amid luxurious shade trees and quiet paths, she lies smiling, just at the base of the mountain called "Royal." In one of these buildings repose the ashes of her greatest son, Sir William Osler, surrounded by the incomparable "Bibliotheca Prima." Hereabout in olden days lay the original settlement of the Indians called Hochelaga, first visited by the intrepid Cartier. Above the university campus on the higher slopes of the mountain are erected the noble buildings of the Royal Victoria Hospital, built by the munificent gifts of two of the university's most constant friends and supporters, Lord Strathcona and Mount Royal, and Lord Mount Steven, and since added to by the gifts of many other loyal citizens and friends. In the city below, nearer the broad reaches of the St. Lawrence, on whose waters so much of human weal and woe has transpired during four centuries, lies the mother of them all, the old Montreal General Hospital. And she, too, smiles as from her vantage point of

many years she contemplates her offspring on the higher ground. Though well past the century mark she still carries on with the best of them — the old "Montreal General," still smiling.

You hear that dame laughing? — You think she's all fun;
But the angels laugh, too, at the good she has done;
Her children laugh loud as they troop to her call,
And the poor man that knows her laughs loudest of all.

(This was written by Oliver Wendell Holmes about a *boy*, — not a *dame*, — but I know you will forgive the adaptation.)

Edward W. Archibald, — surgeon, forward-looking investigator, true son of your beloved alma mater, throughout whose lifework the head, the heart, the hand have all been present in a most happy conjunction, — the Boston Surgical Society bestows on you this Henry Jacob Bigelow Medal with its highest appreciation and love.

ADDRESS FOLLOWING THE PRESENTATION OF THE HENRY JACOB BIGELOW MEDAL

EDWARD W. ARCHIBALD, M.D.*

MONTREAL

IN coming here tonight to receive this great honour, and to offer a very inadequate quid pro quo in the shape of the somewhat rambling remarks which are to follow, I feel that I am returning to visit both friends and — relations! Such a claim from a far Northerner demands explanation, and that I can give only by relating a small part of my family history. I leave out of the account the origin in Stirling, the migration in the early seventeenth century to North Ireland to take up land confiscated from the Irish and offered freely by James I to his English Puritans and his Scotch Presbyterians, both very troublesome; James hoping thereby to get rid of the lot through three-cornered fights between these and the evicted Irish. I pass over the hundred years in and near Londonderry, and the migration to New England of both paternal and maternal ancestors, about 1740: and my own narrow escape when, on the voyage out, the lad Samuel Fisher, of the maternal side, was chosen by lot to be eaten by the famished crew during a long calm, but was saved by the appearance of another ship while he was preparing to meet his God. And I come to their settling in Londonderry, New Hampshire, an hour's ride in these days from this place, the Fishers remaining and the Archibalds soon moving north to Truro, Nova Scotia. Matthew Archibald then comes down on a visit to Londonderry, and takes back with him his bride, Janet Fisher.

There, then, you have it — the relationship! May it add something to the welcome you have already given me! It is told for that purpose only. But there is more — on the side of friendship! When in 1775 the Revolution broke out, and the "embattled farmers" made their first stand, the people of Truro and the neighboring towns of Onslow and Londonderry, in Nova Scotia, immediately declared themselves at one with the revolting colonists, and we find an "undertaking" signed by practically all the citizens, and running as follows:

> In consequence of the resolution of the Hon. Continental Congress, and to show our determination in joining our American brethren in defending the Lives, Liberties, and Properties of the Inhabitants of the United Colonies: We, the subscribers, do hereby solemnly engage and promise that we will, to the utmost of our power, at the risk of our Lives and Fortunes, with arms, oppose the hostile proceedings of the British Fleets and Armies, against the United American Colonies.

And the first three names in the list of signatures were Arthur, Robert and John Archibald, while the fourth name was Samuel Fisher. Like Abou ben Adhem, whose name on a celebrated occasion "led all the rest," so here the name of Archibald was similarly placed. Perhaps I ought not to suggest that on both occasions the list was arranged in alphabetic order. Yet, even so, I do not hesitate to claim something on this score, too, of your friendship and of your indulgence. So I stand here, thanking you in the most heartfelt way for this honour, and extending from Canada a hand of cousinly affection.

Humour, I fear (if this is humour), may be out of place in an address of this sort, yet I can hardly refrain from appropriating and adapting to the present circumstance a joke of my old friend and fellow townsman, Stephen Leacock. If I should do so, I would call your attention to these facts: that Boston is the hub of the universe, the Boston Medical Library is in the centre of Boston, the John Ware Hall is in the centre of the Boston Medical Library, and the medalist stands here in the centre of John Ware Hall. *Quelle pensée solennelle!* Yet the natural elation inspired by such a thought is sadly tempered by apprehensiveness; for I know what the travail of the mountain is about to bring forth.

*Professor of surgery, McGill University.

After these introductory remarks of a deprecatory and conciliatory nature I venture upon the subject of the address. Need I say that I found it difficult to choose a subject? Not from any *embarras de richesses;* rather from poverty of ideas and paucity of knowledge. Dr. Quinby was so good as to suggest that I might discuss the early history of Canadian surgery, or perhaps the subject of the surgical treatment of pulmonary tuberculosis. With the latter I confess myself to be fairly well fed up, an opinion with which most of my friends (and all my enemies) must be entirely in accord. But with the former idea I played for a while, until, happening to make the suggestion at dinner to the family circle, one member of the younger *irreverentsia,* retaining only the phrase "early Canadian surgery," ejaculated, "Ho! scalping, I suppose!"—a remark which gave me pause. I decided against the idea, because, although Canada might well claim priority in the operation of being scalped, having been settled by Whites a good two or three generations before transatlantic shipping was instituted with some regularity by the *Mayflower,* you would nevertheless be quite as familiar with that form of early surgery as ourselves.

Giving this up, my thoughts turned perhaps naturally to Henry Bigelow's early studies in Paris; for I too, as a student, went to France for a winter, although Montpellier was my chosen university, and I finally decided that this particular phase of Bigelow's life, and the possible influence of French education on his later development, might also interest you.

Let me, then, endeavour to trace, though only in its outlines, the state of medicine and of medical teaching in France during the period of the first half of the nineteenth century; we may then be able to guess what profit the eager and critical spirit of Henry Bigelow might draw therefrom.

There was a renaissance in medicine, as in art and literature; but inasmuch as medicine is a science rather than an art, its renaissance had to await the discovery of those laws of nature upon which depend medical science, that is, the normal functioning of the bodily organs and their derangements resulting from disease. It had to await the development of physiologic knowledge, even after anatomic knowledge had been acquired. It needed time for the discovery of the circulation of the blood, the functions of the various systems and the invention of instruments of precision, such as the microscope. It had also first to get rid of mediaeval authoritarianism in that sort of totalitarian state of medicine, which was characterized by "systems" and "theories" unsupported by anything but the dogmatic beliefs of the ancients. It had to await the rise of inductive reasoning, and the application of the inductive system to the phenomena of disease. In art nothing of this was needed. In philosophy it was largely a return to the Greek philosophers. In medicine the true renaissance hardly began before the later years of the eighteenth century, with John Hunter; although Harvey one hundred years earlier had prepared the way with his discovery of the circulation of the blood. And the rebirth which began then has not yet attained maturity. The science of medicine will still add cubits to its stature.

The cardinal fact in John Hunter's life was that he accepted nothing on the mere voice of authority, but must prove all things for himself. To that end he relied upon the observation of facts, and facts alone. Thirteen thousand specimens in the Hunterian Museum of the Royal College of Surgeons in London, all prepared and mounted by his own hands, attest this statement. He, the first, called in the experimental method to support inductive reasoning. He died in 1793.

Yet, up to the time of Laennec in the early years of the nineteenth century, notwithstanding a considerable number of advances made in our knowledge of physiology, it is fair to say that, on the whole, medical knowledge was confined to the things of the body surface; things that one could see and touch. Laennec's invention of the stethoscope, early in the 1800's, combined with his post-mortem observations, first revealed the interior of the thorax to the mind of the physician. With him came the new principle of the correlation of clinical facts with dead-house observations, which replaced the older teachings based upon theory and unregulated fancy, and built up gradually in the course of centuries by those who did not and could not know. This fundamental change in the principles of acquiring knowledge was the real renaissance in medicine, and from it there flowered forth all those discoveries of the last hundred years and more which now beautify and glorify the demesne of scientific medicine. I think of that renaissance of clinical medicine as being primarily due to Hunter, and then as being developed chiefly by the French school of the first quarter of the nineteenth century, in particular by Bichat, Laennec and Louis. The English clinicians quickly followed with the celebrated Guy's Hospital group, Bright, Addison and Hodgkin, and ten years later with the Dublin school of Stokes, Cheyne, Corrigan and Graves. Physiology about the same time made rapid advances under Sharpey, Flourens and Magendie. The microscope, enormously improved by the achromatic lens invented by the wine and spirit merchant who was Joseph Lister's father, came in at this point to enlarge inconceivably the field of observation, and to give us within the scant space of two decades the great work of Virchow on

cellular pathology. Medical knowledge was expanding at a pace which in the eager eyes of youth must have been thrilling. But for the surgeon that thrill of new knowledge, to one endowed with natural sensibilities, must have been sadly tempered by the pain he was obliged to inflict, and the all too frequent mortal infection of his wounds.

Into this setting stepped that ardent spirit, Henry Jacob Bigelow. He might well bless the hour of his birth and the day that brought him forth, for he lived in great times. It was a period of great achievements, of unlimited opportunity for discovery, and on his own stage he played a great part. What manner of youth, then, was he? It might ill become me, a stranger, to attempt to tell you of his native city, including perhaps a few who knew him in life, what manner of man he was. Yet, for the sake of the development of my theme, you will surely forgive me if I extract from the biographic records, written by those who knew him best, a few details which may perhaps serve to illustrate the general lines of his character and the effect which his studies in France might well have had upon his development as a surgeon. I have diligently read all the available material concerning him, or at any rate all which was accessible to me, and have read what he himself wrote. Unfortunately, hardly any of his letters home from France seem to have been preserved; but in the *Memoir* of his life, and in the proceedings of the memorial meeting of the Boston Society for Medical Improvement in November, 1890, there is sufficient record written by his contemporaries from which to trace a picture. From this record I draw freely.

He was born in 1818, just four years after Sir James Paget, to hear whose lectures on pathologic anatomy he made (twenty-five years later!) a weekly journey from Paris to London. He came of very fine stock. His father gave him great physical and mental vigour, and his mother strength of character and capacity for work. He was a handsome boy, with red cheeks, blue eyes, light hair and a goodly figure. His disposition was pleasant and companionable, brimming over with spirits, ever on the alert. He was "the light and delight of his home, the most charming, affectionate, gay and cheery son and brother." He was expert in dancing and swimming, and in the gymnasium. He was born with the investigative spirit, and this was backed up and made concrete by the gift of mechanical ingenuity. He was "fertile in contrivances of all sorts." And he had perseverance to boot: before arriving at the final perfect model of his lithotrite he had worked through and discarded a dozen models, each embodying some improvement on its predecessor. And, strangely enough, set over against this mechanical turn of mind, he loved music, was a member of the Pierian Sodality (a musical club) and played the French horn. Cheever said in the *Memoir* that there was more of the Latin, or French, in him than of the Anglo-Saxon. He was *vif* and *spirituel;* but he also had Anglo-Saxon indomitableness. He was a great lover of nature, and made collections of various animals; but it had to be always a particular species and the collection the finest anywhere. His spirit was one of great independence. As a lad he made his decision to be a surgeon. James Jackson remonstrated, and urged him to follow in his father's footsteps and be a physician. Henry simply replied, "I'll be damned if I won't be a surgeon!" Dear old Oliver Wendell Holmes in summarizing the qualities of Henry Bigelow, adapted slightly the words of Horace in the *Ars Poetica* describing Achilles. No more than one line of four words! But fully interpreted, how revealing! These words were:

Impiger, iracundus, inexorabilis, acer.

Only he altered, very justifiably, *iracundus* to *jucundus*. Now, the only one of these words that I did not know — and I say it to you with a justifiable pride (coming from the barbarian North) — was *impiger*. My old Latin dictionary, I may tell you, was *not* in the cellar, buried under the books of my youth, Brachet's *Grammaire Historique,* Kant's *Critique of Pure Reason* and various theological works. It was upstairs, close by, at hand. Let me give you, unnecessarily of course, what I found in it. First, *"Impiger, gra, grum,* adj.: not indolent, diligent, active, quick, unwearied, indefatigable, energetic."

Horace's second word was *iracundus,* "quick to wrath," which Holmes changed to *jucundus,* which has the significance of good humour mixed with joyousness. He may have spoiled the meter slightly, but the substituted word was the truer word.

The third word was *inexorabilis.* The root verb *oro* means "I plead, argue, pray, beg, beseech"; and *inexorabilis* may be construed "that cannot be moved by entreaties." Holmes, most ingeniously, paraphrases the word. Nature is depicted as refusing to yield up her secrets, but Henry Bigelow is "inexorable" in his demand that she shall do so. This is not quite Horace's meaning as applied to the old hero with the heel, but we are grateful nevertheless to Holmes for the ingenious turn.

Now when we come to the last word, *acer,* we are on sure ground and can let ourselves go. My good old Lewis and Short thus translates the word: as applied to the intellectual qualities it means "subtle, acute, penetrating, sagacious, shrewd"; and as applied to moral qualities we find it means "ac-

tive, ardent, eager, spirited, brave, zealous." Now, I ask you, could one find a better summary of Henry Jacob Bigelow's character?

But I should like to add another word to these four, at whatever expense to meter or any other consideration. That word is *misericors*. This time I hunted through the other half of the dictionary to find the Latin for tender-hearted, and I have the support of our professor of classics in the choice of *misericors*. Bigelow was indeed tender-hearted. His two great qualities, as I have read, were untiring devotion to his patients and his reluctance to give pain. Dr. Hodges said that his tenderness toward children was always noticeable, while his gentleness with dumb animals found its complete expression in "a strenuous opposition to repetitive, unavailing or incompetent vivisection." "No suffering invalid ever found him rough or thought him brusque"; and we should note also his immediate, enthusiastic and forceful support of Morton's ether anesthesia in 1846, when he was only twenty-eight years of age and still the youngest surgeon in Boston.

Henry Bigelow studied medicine with his father. In those days the requirements as to study leading to the degree of M.D. were somewhat simpler than now. Students entered their names with practising physicians from whom, for a period of three years, they were supposed to receive medical instruction in various ways. During these three years two courses of lectures of four months each were to be attended at some regular medical school. That was all. But young Henry was more fortunate than many in that he had the constant companionship and instruction of his father and his father's friends, particularly of two, James Jackson and Oliver Wendell Holmes. With the latter he took his anatomical course at Dartmouth. From 1838 to 1839 he was house surgeon in the Massachusetts General Hospital. On account of certain pulmonary symptoms he went to Cuba for several months in the fall of 1839, and thence, in company with his friend Dr. Samuel Cabot, sailed for Europe, coming home to take his medical degree in the summer of 1841. Yet he returned almost immediately to Paris, the acknowledged centre of medical instruction at the time, in order to pursue his studies.

I have already indicated very briefly the main lines along which medical science was developing in France. It remains to sketch — for one can here do no more than sketch — the professional and educational *milieu* in which young Bigelow now found himself. It was the year 1842. Internal medicine had but lately escaped from the trammels of the theorists, blind children of the Scholiasts, still stuck at the level of Galen, Hippocrates and the Arabs, learned in logic and dialectic but not in diseases. At the end of the eighteenth century they were still arguing about the humours and the elements and the temperaments and the organs noble and ignoble; about the relations of the elements with the four seasons, the four stages of life and the four humours. In contrast with this, surgery, having to do with things visible and tangible, for which it could often bring quick and obvious cure by operation, had long been making progress, inasmuch as it needed only a solid knowledge of anatomy and greater courage based upon increasing experience. If the people demanded a sign, that is, a miracle, the surgeon might often satisfy that demand; but the physician rarely. Yet so long as surgery, before the light of antisepsis dawned, dared not invade the cavities of the body, its progress had to remain limited to the external tissues. Nevertheless its field broadened steadily during the later years of the eighteenth and the early years of the nineteenth centuries. Take, for instance, the clinical lectures of Dupuytren during the years from 1820 to 1835. They fill six octavo volumes, running from five hundred to seven hundred pages each. I should weary you if I went into detail, but the point is that the lesions treated were all external.

From all this we must not conclude that Britain and America were behind in surgery. Paris boasted of great names, but in England was there not John Hunter, greater than all his successors, and were there not men like Astley Cooper, Abernethy, and then Syme, Liston, Fergusson; and in America did not John Warren (besides helping to throw the tea overboard in your harbour, when he was only twenty) perform one of the first abdominal sections recorded in this country, the opening and evacuation of a dermoid cyst in the left hypochondrium, with recovery of the patient, and this in 1785 at the age of thirty-two; and follow it up with a successful amputation at the shoulder joint? And I need do no more than mention the name of John Collins Warren, who, from 1806 on, did bold and brilliant surgery in this city, among other things conducting the first operation in 1846 under ether anaesthesia. John Mason Warren, seven years older than Bigelow, went to Europe in March, 1832, studied in the clinics of Astley Cooper, Charles Bell, Syme and Liston, and then proceeded to Paris, where he studied surgery under Dupuytren, Lisfranc and Roux, and medicine under Louis. His companions in Paris were Jackson, Bowditch and Holmes, with Gerhard, and Pepper of Philadelphia — a brilliant group. He, too, after his return in 1835, did splendid surgery. So that Bigelow, when he returned to Boston in 1844, found a surgical environment well established

and up to date, and affording that stimulus of competitive work which always leads to progress.

But if surgery in France was not materially in advance of Britain, wherein lay the attraction of Paris for our young Americans? The answer is simple. It was not alone, or chiefly, the enormous clinical material available to the French professors for teaching, or a possible superiority in clear and logical exposition; it was much more the organization and the constitution of the Paris Faculty of Medicine, state-controlled and state-paid, and wonderfully adapted for the education of both undergraduates and postgraduates. Perhaps an even stronger attraction was the extraordinary opportunity for postmortem examinations. In France it was the law that an autopsy might be done on every patient dying in the city hospitals. In Britain and America postmortems were hedged about with difficulties, and were consequently rare. Even anatomy in these countries suffered seriously from a lack of subjects. Resurrectionists went in peril of their lives. Bootlegging — obsolescent word, yet to memory dear! — bootlegging of bodies became a regular trade. Burke and Hare committed murder for it. Anglo-Saxon feeling among the laity could not bear the thought of "dissections"; but in France one could buy a cadaver easily and legally for as little as ten francs. France had obviously the jump on the rest of the world. It is doubtful if anywhere else than in France could Laennec have confirmed the observations made through percussion and auscultation by a regular examination of the intrathoracic organs after death in so many cases; or if Louis could have written his illuminating monograph on typhoid fever. Small wonder, then, that with the new spirit of inductive reasoning on the basis of facts observed, these magnificent opportunities of collecting the facts put France far ahead of other countries, and drew students from all parts of the world. It was in Bigelow's character that he always sought facts, but particularly new facts, and that he could not rest until he had exhausted any subject that caught his interest, and had added something of his own to it. In this sense we cannot doubt that he found great mental satisfaction in Paris. But it may be doubted that he found there anything in the field of surgery so new and so illuminating as the pathbreaking work of Laennec in diseases of the heart and lungs, or Louis's numerical method, which had attracted so strongly his internist friends from Boston, New York and Philadelphia. Doubtless he saw new operations and the boldest, most skilful kind of work; but the really new thing that attracted him, we may believe, was not operative surgery but the young science of surgical pathology. The microscope was just beginning to be used by the younger men who had surgical ambitions, and Bigelow discovered that the one who could teach him most in macroscopic and microscopic surgical pathology was young James Paget, in London, curator of the museum of the Royal College of Surgeons, and recently appointed lecturer in physiology at St. Bartholomew's Hospital. Such was Bigelow's love of fundamental knowledge, and his vision of the future path which scientific surgery must tread, that he forthwith undertook the long journey to London once a week in order to follow James Paget's lectures. Only ten years previously Holmes had complained bitterly of the diligences that took thirty-six hours from Boulogne to Paris; but the railway, I fancy, must have been opened in the intervening time. Even so, what a journey to take regularly each week! Since the time of Paget, who for close on ten years, worked among pathologic specimens before securing any hospital appointment, every great surgeon has recognized and has early secured that preliminary foundation in the fundamental sciences.

History shows that leadership in medicine goes to that country which provides best for instruction in the *science* of medicine. Italy and France from the twelfth to the seventeenth centuries drew students from all Europe to Salerno, Bologna, Padua, Montpellier and Paris. Britain drew them in the late seventeenth and eighteenth centuries to London and Edinburgh. France, even through the fermenting years of the Revolution and the young Republic, and still more during the peace of the Bourbon Restoration, drew them to Paris from 1800 to 1850. A short generation later and Germany stole the show from Paris, because, recognizing this fundamental fact, she offered for the world's use a great number of laboratories in all branches of medicine, magnificently equipped and splendidly led. This lasted up to the Great War. But with Germany's defeat in war, and with, lately, the new spirit that guns are more necessary than laboratories, let alone butter, the student is turning again from her to the Anglo-Saxon countries, and to America not less than to Great Britain, because he finds there superior opportunities for acquiring knowledge, not in the *art* but in the *science* of medicine. So it was that Bigelow saw the great opportunity of his time; his own enquiring spirit drove him to profit by it, and the new fundamental knowledge thus acquired gave him immediately a standing at home which led to quick and lasting success.

But a few words of further detail concerning the organization of medical teaching in Paris seem necessary and may be interesting.

The Royal Academy of Surgery of Paris was suppressed during the Revolution, the *sans culottes* spirit having no use for science; but within a year and a half, as Napoleon's army was in need of

surgeons, science was again recognized, and the École de Médecine was re-established in Paris, Montpellier and Strasbourg. In the new foundation the whole teaching corps was placed under a single administration of the government. The advantage was that this created a complete barrier against the intrusion of unqualified pretenders into the field of teachers, and that it demanded the adoption of a long period of instruction on the part of the students. It meant also a *unified* system of instruction for all universities in France. The public was assured that the individual doctor to whom they entrusted their lives had received a proper medical education; and the schools were prevented from underbidding each other with a view to attracting a large number of students.

In the *American Journal of the Medical Sciences* for May, 1831, there appeared a long article on the state of the medical schools of Paris, and the author suggested the advantages to be reaped in the United States were a similar plan to be adopted and the direction of public education placed in proper hands. Under such a plan "we should see all the charlatan teachers, with which our country is fairly deluged — all those cheap schools which have nothing to recommend them but the low price at which they sell their scanty instruction — vanish and give way to schools established on a more extensive, rational, useful plan."

At the École de Médecine, the student before receiving his diploma had to furnish certificates proving that he had complied before entering upon the study of medicine with certain prerequisites; for example, that he had obtained the degrees of Bachelor of Letters and Bachelor of Science. The first required knowledge of Greek literature, Latin eloquence, Latin poetry, French eloquence, French poetry, philosophy, history of philosophy, ancient and modern history and ancient and modern geography. And for the second, the candidate must have passed satisfactory examinations in mathematics, natural philosophy, chemistry, zoology, botany and mineralogy. Only after all this could he commence the study of medicine, and even then only after providing a certificate of correct and moral behaviour and furnishing a security. In the intervening hundred years we have probably attained a higher level of humanity in the practice of the profession; but the requirements of our premedical course have remained below those of France in 1837.

The term of medical instruction was four complete years. There were two semesters, winter and summer. The summer holidays occupied only September and October. The price of medical education amounted to one thousand francs, and in addition one hundred francs for the seal of the diploma, and three francs for the use of the gown worn at the time of examinations.

Sixteen divisions of general medicine were taught, each with its own professor. The writer of this article concluded as follows:

> The plan in question is so far superior to that in force in our schools, the effects of this superiority are so evident to everyone who has been able to make the comparison between the graduates of both countries, and who is not biassed by prejudices, that, were we called upon for advice by a young man about to commence the study of medicine, and were we able to select the place where he was to prosecute his studies, we would unhesitatingly give the preference to the schools of France over those of our own or of other countries. We sincerely hope that the eyes of our countrymen will, before many years have elapsed, be open on this important matter, and that the necessary reforms will be made in the plan of instruction actually in force among us.

It needed some little time before this pious hope was realized.

Hospital organization was magnificent. There were twenty thousand beds in the Paris hospitals available for the instruction of students, and the type of instruction was almost the same as that which now prevails.

The great Dupuytren had died in 1835, but his rival, Lisfranc, was still active at La Pitié. This hospital had six hundred beds. Lisfranc had a brilliant career. His *Traité de Médecine Opératoire* was published in 1845, and embodied the surgical technique of the first third of the century. Roux, of the Hotel Dieu, was also a great surgeon. He was the first to realize the operative cure of perforations of the palate, and the first to use, in France, Hunter's operation for aneurysm. Récamier was the medical colleague of Dupuytren at the Hotel Dieu, but he was really a surgeon who had strayed into the field of medicine. He was the first in France, or, I believe, anywhere, to do a successful vaginal hysterectomy, which he finished in twenty minutes. He was a close friend of Laennec and was extraordinarily modern in his ideas concerning medical treatment. He banished leeches; he would have none of the usual starvation diet; poultices on wounds he abhorred; and he gave cold baths for typhoid fever. Altogether he was one of the most original of the surgeons in France. Velpeau was at La Charité, which had five hundred beds. He was apparently one of the most humane of the Paris surgeons. He opposed unnecessary operations on the sole, but sufficient, ground that they were inhuman. He was a prolific writer both on surgery and on surgical anatomy. His honesty and his humanity must have attracted Bigelow. Then there were Amussat and Civiale, both specializing to some degree in urinary diseases. Civiale is generally credited with the invention of the first practical lithotrite, and

one imagines that Bigelow must have seen its disadvantages, and must have already conceived the idea of improving the instrument, which, as we all know, he later perfected.

Space fails me to do more than mention other prominent surgeons of this period. There was Gustav Monod, the first of a long line of famous surgeons in France. Then there were Malgaigne, Sedillot and Ricord, all names that stand out in the history of surgery. Malgaigne was a professor in the grand style, and one of the most cultivated men in the profession. He must have made a special appeal to young Bigelow. Then there was Nélaton, he of the rubber catheter and the porcelain probe, with which he found the lead bullet in Garibaldi's foot.

Orthopaedics attained first in France the dignity of a specialty, although orthopaedic surgery had long been practiced. In 1741 appeared the first treatise on this subject, by Audry, of Montpellier. Guérin, of Paris, orthopaedist and surgeon to the Enfants Malades, was one whom, Dr. Osgood tells me, Bigelow followed particularly. This hospital had five hundred and sixty beds. From Guérin, Bigelow probably learnt subcutaneous tenotomy, a great operation in preantiseptic days. From him also, very probably, came to Bigelow the impulse to write his first major thesis "Orthopaedic Surgery," which won him the Boylston Prize, and which may have led on to his original work on dislocation of the hip.

In imagination one may reasonably think of our young Bigelow in Paris — he of the keen observation, soundness of judgment, independent spirit and the pitiful heart, hating unnecessary pain, gentle and compassionate to the sufferer — one may think of him as avoiding the brutal surgeons like Lisfranc, as he would surely have avoided the most brutal, Dupuytren; frequenting the clinics of the humane, and equally able and more cultured, like Velpeau, Malgaigne and Récamier; turning in other hours to the new and brilliant clinical and pathological lectures of the great Louis, and Louis's friends, Andral and Chomel; listening with distaste to the Gallic cynicisms of Ricord, chief of the Hospital of St. Louis for venereal cases; and taking frequent refuge at the orthopaedic division of the Enfants Malades under Guérin, where surely, among little children, there reigned as humane a surgery as was possible in those preanaesthetic days. Doubtless his natural skill in the handicraft of surgery was augmented by watching, probably in London as well as in Paris, the masters of the art at work. His colleague, Cheever, said after Bigelow's death, concerning his operations, that "as a surgeon, in method, he was a pupil of the French school. To ingenuity he added dexterity, and to dexterity grace. He was alert, cool, practical. Precise in touch, supple in movements, he added the polish of the finished artist to the nonchalance of the experienced operator. To see him operate was to recognize a master."

Here, gentlemen, I must stop. It is unfortunately impossible, in the lack of anything definite from Henry Bigelow's own pen, to trace in detail the influence of any one Paris surgeon, or of many, upon his mind and his surgical training. But the general effect of French teaching and example, not only in surgery but in his attitude toward the art of living, was obviously great. Dr. Cheever, again, said of him in 1890 at the memorial meeting that he was of Latin rather than Anglo-Saxon type. The logic of the French, who are great "realists," and their ability to classify and clarify a subject and set it forth concisely and simply were also his qualities. In addition, he had that love of living gracefully and in a cultured way which the French know so well. But above all — though I repeat myself — he found in France and with Paget in London the means of satisfying the demands of his intellect for knowledge, especially that fundamental knowledge of physiology and pathology upon which, as his clear mind saw, had to be based any real progress in the science of surgery. This was perhaps the chief thing that France did for Henry Jacob Bigelow.

PARA-AMINOBENZENESULFONAMIDE AND ITS DERIVATIVES IN THE TREATMENT OF BETA HEMOLYTIC STREPTOCOCCUS INFECTIONS OF THE MIDDLE EAR AND MASTOID

Report of Six Cases in Children

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FOLLOWING the report of Long and Bliss[1] on the treatment of infections caused by beta hemolytic streptococci with para-aminobenzenesulfonamide and its derivatives, other papers concerning the use of these drugs in the treatment of similar types of infections have been published.[2, 3, 4, 5] The present report deals with the use of para-aminobenzenesulfonamide and its derivatives in the treatment of patients with complications of middle-ear and mastoid infections caused by beta hemolytic streptococci. This includes 3 patients with meningitis, 1 patient with sterile meningitis, 1 patient with perisinus abscess and septicemia and 1 patient with postscarlatinal mastoiditis.

Case 1. K. M., a white male, aged 8 years, was admitted to the Children's Hospital, March 19, 1937, complaining of a right ear which had been discharging for 1 month and pain for 3 nights prior to admission. The otorrhea had been profuse and constant until 3 days before admission, when it began to decrease. During the week before admission intermittent frontoparietal headache and postaural pain had been prominent symptoms. There was no nausea or vomiting. No chills had occurred. A few hours prior to admission the patient had epistaxis. The temperature was 101.6°F. The patient was a poorly nourished boy, alert, apprehensive, but apparently in no pain. The eye grounds were normal. The right ear was pushed downward and forward by a small, nonfluctuant, postaural swelling. Slight tenderness was elicited over the tip of the mastoid. The right drum showed redness, loss of landmarks, posterior bulging and an anterior perforation through which a small amount of purulent material was escaping. Except for some crusts in the nares and several carious teeth, the remainder of the examination was negative. The blood hemoglobin was 80 per cent (Sahli); there were 15,800 leukocytes per cu.mm., 70 per cent of which were polymorphonuclear cells. The urinalysis was normal.

The day following admission the temperature was 100°F., and the patient no longer complained of pain in the ear. In 36 hours the temperature was normal, and he seemed very much better, but the discharge from the right ear had increased. Four days after admission a simple right mastoidectomy was done. The mastoid cells contained free pus and granulations, and in many places destruction of the cell walls was noted. The dural plate was intact and was not disturbed. A small area of the sinus, near the mastoid tip, was exposed and appeared normal. The wound was left open. Beta hemolytic streptococci were cultured from the pus in the mastoid. The postoperative course was uneventful for 8 days. At this time the boy developed an upper respiratory infection with fever, cough and nasal discharge, from which he recovered in 5 days.

On April 12, the 19th postoperative day, the temperature rose to 103.8°F. at 4 a. m. The patient had no complaints. Examination of the lungs was negative. Urinalysis was negative. The mastoid wound was clean and nearly healed. Examination of both middle ears and the nasopharynx was negative. Neurologic examination was negative. The following day the temperature was normal during the morning and afternoon. There were 31,800 leukocytes per cu.mm. in the blood. The patient's appetite was good, and he had no complaints. At 8 p. m. the temperature was 104.6°F. The patient was oriented and was not irritable. Careful physical and neurologic examinations were negative except for slight engorgement of the vessels in the fundi.

On April 14 the temperature was 104.6°F. at 8 a. m. The neck was definitely stiff, and Kernig's sign was positive. The vessels of the fundi showed distinctly more engorgement, and the margin of the left disk was blurred. The patient complained of slight headache and seemed drowsy, but was oriented and not irritable. He had not vomited. A lumbar puncture was done and slightly cloudy cerebrospinal fluid, which contained 1160 leukocytes per cu.mm., was obtained. Gram-positive cocci in chains were seen in a stained smear of the fluid, and beta hemolytic streptococci were obtained on culture. The mastoid wound was reopened and the bone covering the dura of the middle and posterior fossae was removed extensively, but no visible focus was found. Para-aminobenzenesulfonamide was administered intrathecally and subcutaneously. Later prontylin was given orally. Rapid improvement occurred. The cerebrospinal fluid rapidly became normal and the patient was discharged May 10. (See Table 1.)

Case 2. P. M., a white female, aged 5 years, was admitted to the Children's Hospital, May 5, 1937. The chief complaints were headache, vomiting, drowsiness, stiff neck and discharge from the right ear of 1 day's duration, preceded 2 weeks before by pain in the ear. The temperature was 103.6°F. The patient was a well-developed, poorly nourished, acutely ill child lying quietly in bed on her left side with her head slightly retracted. When roused she was disoriented and irritable, and resented any attempt to change her position. The right ear canal contained a large amount of thin yellow pus. The drum showed an adequate anterior perforation through which a pulsating stream of pus flowed. There was no postaural swelling or thickening. It was impossible to determine the presence or absence of mastoid tenderness. The fundi were normal. Kernig's sign was positive and marked cervical rigidity was present. X-ray of the mastoids revealed diffuse clouding of the right with no

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evidence of destruction, and neither petrous tip showed any evidence of invasion. Urinalysis was negative except for the presence of from 5 to 10 leukocytes per high-power field in an uncentrifuged specimen. Examination of the blood showed 67 per cent hemoglobin (Sahli), and 3,640,000 erythrocytes and 12,700 leukocytes per cu.mm., 83 per cent of the latter being polymorphonuclear cells.

A lumbar puncture on admission yielded cloudy cerebrospinal fluid under normal pressure. The globulin content was increased and sugar was absent. The fluid clotted before the leukocytes could be counted. No bacteria were seen in a stained smear, but beta hemolytic streptococcus was cultured from the fluid. The lumbar puncture was repeated and para-aminobenzenesulfonamide was injected intrathecally and subcutaneously. The right mastoid was opened and wide exposure of the dura of the middle and posterior fossae made. The mastoid cells, some of which showed destruction of the bony walls, contained pus and granulations, and beta hemolytic streptococcus was cultured from the pus. No gross evidence of extension to the meninges was found. Following operation the patient received para-aminobenzenesulfonamide intrathecally and subcutaneously. Later, prontylin was given by mouth. The fever gradually subsided, the cerebrospinal fluid returned to normal, the meningeal signs disappeared and an uneventful convalescence was followed by discharge from the hospital on May 26, 1937, 3 weeks after admission. Subsequent examinations at weekly intervals during the following 6 weeks were normal. (See Table 2.)

Table 1. *Summary of Case 1.*

DATE	PABS Intraspinally	PABS Subcutaneously	PRONTYLIN Per Os	CEREBROSPINAL FLUID	REMARKS
1937	*cc.*	*cc.*	*gm.*		
April 14	25	200	—	Lumbar punctures at 10 a. m., 3 p. m. and 9 p. m. 1160 leukocytes per cu.mm. in fluid at first lumbar puncture. Sugar absent. Beta hemolytic streptococci cultured from cerebrospinal fluid at each lumbar puncture and observed in smear.	T. 104.6°. Neck stiff. Kernig positive. Investigation of right mastoid. 350 cc. 10% dextrose solution intravenously postoperatively. Took fluids by mouth. Blood erythrocytes 5,500,000. Blood leukocytes 40,000.
	35	200	—		
April 15		200	4	Cerebrospinal fluid appeared less cloudy. Culture sterile.	T. 101.6°. Took fluids well. Seemed better. Well oriented. No complaints. Neck less stiff. Kernig sign positive. Blood erythrocytes 5,000,000.
April 16			4		T. 102°. Took nourishment and fluids well. Neck very slightly stiff. Kernig sign barely perceptible. Blood erythrocytes 4,200,000. Blood leukocytes 19,400. Transfusion 250 cc. citrated blood.
April 17			4		T. 102°. Neck not stiff. Kernig sign negative. Appetite good. No complaints.
April 18			4		T. 101°. Appetite good. Sat up in bed reading comics. No complaints.
April 19			4		T. 99.8°. No complaints. Wound healing with very little discharge.
April 20 to May 10			4 daily until April 23		Except for a mild attack of varicella on April 30 the remaining time in the hospital was uneventful. Discharged May 10.

PABS — 0.8% para-aminobenzenesulfonamide in physiologic salt solution.
T. — Highest temperature of day.

Table 2. *Summary of Case 2.*

DATE	PABS Intraspinally	PABS Subcutaneously	PRONTYLIN Per Os	CEREBROSPINAL FLUID	REMARKS
1937	*cc.*	*cc.*	*gm.*		
May 5	30	250		Cloudy. Globulin positive. Sugar absent. Clotted, no leukocyte count. No bacteria seen on smear. Beta hemolytic streptococci on culture.	T. 103.6°. Lumbar puncture and right mastoidectomy. Blood culture sterile. 200 cc. 10% dextrose solution intravenously.
May 6		300 250	1.0	Cloudy. Globulin positive. Sugar present but diminished. Clotted, no leukocyte count. Culture sterile.	T. 102.6°. In a. m. seemed better. Answered questions and took fluids well by mouth. Had a slight nosebleed. Worse in p. m. with mild delirium and refused to take fluids. Cyanosis of lips and nail beds. Transfusion 200 cc. citrated blood. Neck stiff and Kernig positive.
May 7	30		2.6	Culture sterile.	T. 103°. Delirium disappeared in a few hours. Seemed better. Took fluids and semisolids well. Neck less stiff. Kernig positive.
May 8			2.6	Cloudy. Globulin positive. Sugar present. 1800 leukocytes. Culture sterile.	T. 104°. Seemed better. Less cyanosis. Slight headache. Appetite good. Frequency of urination with dysuria but urinalysis negative. Very slight stiffness of neck. Kernig diminished. Mastoid wound dressed.
May 9			2.6		T. 101.4°. Very much better. Mentally clear. Sat up in bed and read comics. Appetite good. Neck not stiff. Kernig negative. Wound dressed.
May 10			1.3		T. 99.6°.
May 10 to 15			1.3 daily		Except for a rise to 103.6° following a blood transfusion on May 11, temperature remained normal. Mastoid wound healed uneventfully. Discharged May 26.

PABS — 0.8% para-aminobenzenesulfonamide in physiologic salt solution.
T. — Highest temperature of day.

Case 3. R. K., a white male, aged 5 years, was admitted to the Beth Israel Hospital, April 25, 1937, with a complaint of left otorrhea of 4 weeks' duration following an upper respiratory infection. The discharge from the left ear through a central perforation in the drum was profuse, thick and greenish yellow, and pulsated. The drum was red and thickened and had lost its normal landmarks. The adenoids were hypertrophied. The remainder of the examination was negative. Urinalysis was normal. The blood hemoglobin was 75 per cent (Tallqvist); there were 4,400,000 erythrocytes and 14,500 leukocytes per cu. mm.

The day after admission a left simple mastoidectomy was performed. Beta hemolytic streptococci were cultured

from the mastoid. Early in the morning following the operation the patient had a sudden attack of nausea, vomiting and headache. It was noted that he fell toward his left side and had a third-degree horizontal nystagmus to the right, with pass pointing to the right. The Kernig and Brudzinski signs were negative, and the neck was not stiff. All packing was removed from the mastoid wound. His condition was unchanged the next day, and the temperature varied between 99 and 100°F. Prontosil and prontylin were started at this time and were discontinued 2 days later. During the next 7 days the child vomited frequently and seemed irritable but had no headache. The nystagmus persisted, but falling to the left side had ceased. On May 7 a swelling of the left cervical nodes was noted, which disappeared following treatment with hot compresses. May 9, or 13 days after the operation, the patient appeared very ill and his temperature was 103°F. He was perspiring profusely, vomiting frequently and complaining of severe headache. A lumbar puncture was done and cloudy cerebrospinal fluid was obtained which yielded beta hemolytic streptococci on culture. A radical mastoidectomy was performed and some destruction of the tegmen tympani was observed. Prontylin was given orally, and the following day the patient received intrathecal and subcutaneous injections of para-aminobenzenesulfonamide. His condition improved steadily, and the temperature remained normal after the 5th day following the second operation. Eleven days after the second operation the patient was entirely normal. (See Table 3.)

Case 3. Beta hemolytic streptococci were cultured from all these specimens of cerebrospinal fluid. The temperature did not remain at a normal level until the fifth day after the drugs were started in each of the cases.

The neurologic signs which had been present in each of the patients at the onset of the meningitis had disappeared by the fourth day in Case 3, and by the fifth day in Cases 1 and 2. Marked improvement was observed in each patient after forty-eight hours of treatment with prontylin and para-aminobenzenesulfonamide. General supportive treatment in the form of parenteral fluids and blood transfusions was given to each patient. At operation no route of extension from the mastoid to the meninges was found in any of the patients. The mastoid wounds healed uneventfully in all patients.

Except for the rapid drop in the blood erythrocytes in Case 1, and cyanosis of the lips and nail beds in all 3 patients, no reactions were noted which might be attributed to toxic effects of the drugs.

Case 4. N. G., a white male, aged 6 years, was admitted to the Children's Hospital on February 10, 1937, complaining of discharge from both ears for 3 weeks following

Table 3. *Summary of Case 3.*

	CEREBROSPINAL FLUID				PABS		PRONTYLIN	
DATE	Culture	Leukocytes	PMN	Sugar	Intrathecally	Subcutaneously	Per Os	REMARKS
1937		*per cu. mm.*	*%*	*mg. %*	*cc.*	*cc.*	*gm.*	
May 9 Preop.	Beta hem. strept.	2500	80	70			3	T. 103. Blood culture sterile. Left radical mastoidectomy.
Postop.	Beta hem. strept.	3000	70	27				
May 10	Sterile	1596	95	45	10	200	4	T. 101.8. Patient appeared better.
May 11	Sterile	176	90	66	12	200	4	T. 102. Transfusion 150 cc. citrated blood.
May 12	Sterile	68	90		5	200	4	T. 100. Cyanosis observed. Transfusion 150 cc. citrated blood. No signs of meningitis.
May 13	Sterile	61	90	70			4	T. 100.2.
May 14							2	T. 99.2.
May 15	Sterile	70	75	57	5		2	T. 99.2. Condition appeared good.
May 16							2	T. 99.2.
May 17	Sterile	67	20	77			2	T. 99.2.
May 18							2	Transfusion 200 cc. citrated blood. Doing well.
May 19							2	T. 99.2.
May 20	Sterile	32	3	74			2	Transfusion 200 cc. citrated blood.
May 21 to 28							2 daily	Making uneventful recovery.

PABS — 0.8% para-aminobenzenesulfonamide in physiologic salt solution.
T. — Highest temperature of day.
PMN — Polymorphonuclear leukocytes.

Treatment was started in Cases 1 and 2 with para-aminobenzenesulfonamide subcutaneously and intrathecally. Prontylin was given orally as soon as the condition of the patient permitted. Case 3 received no subcutaneous or intrathecal treatment with para-aminobenzenesulfonamide until twenty-four hours after the beta hemolytic streptococci were discovered in the cerebrospinal fluid, but he received prontylin orally in adequate dosage during this period. Three lumbar punctures were done the first day in Case 1, one in Case 2 and two in

an upper respiratory infection. The temperature was 101°F. There was a profuse, pulsating discharge through an inferior perforation in each drum. There was no postaural tenderness, but there was some periosteal thickening without fluctuation over the left mastoid. The tonsils were large, boggy and reddened. There was generalized lymphoid hyperplasia in the pharynx. The remainder of the examination was negative. Urinalysis was negative. The blood hemoglobin was 70 per cent (Tallqvist), and there were 3,960,000 erythrocytes and 16,400 leukocytes per cu.mm.

Bilateral mastoidectomy and adenoidectomy were done the day following admission. Beta hemolytic streptococci

were cultured from each mastoid. The temperature rose from 101 to 103° F. during the 2 days following operation. There was no vomiting, and fluids were taken well. The child appeared uncomfortable, but on examination no abnormal findings were noted to explain the rise in temperature. The morning of the 3rd postoperative day (February 15) the temperature was 100°F., and it rose to 104.2°F. in the following 8 hours. Cervical rigidity and a positive Kernig's sign were noted at this time. The blood contained 19,200 leukocytes per cu.mm. A lumbar puncture was done and cloudy cerebrospinal fluid containing 2800 leukocytes per cu.mm., 65 per cent of which were polymorphonuclear cells, was obtained. No bacteria were seen in a stained smear of the fluid and none were cultured from it. Treatment was begun with prontosil and prontylin. The temperature rose to 105°F., 12 hours after the lumbar puncture, then started to fall, and in 24 hours was 96.4°F. Cyanosis of the lips and nail beds was present at this time. The blood hemoglobin was 55 per cent (Tallqvist) and there were 3,250,000 erythrocytes and 7200 leukocytes per cu.mm. On repeating the lumbar puncture the cerebrospinal fluid appeared clear; it contained 440 leukocytes per cu.mm., 2 per cent of which were polymorphonuclear cells, the sugar was 51 mg. per cent, and no bacteria were obtained on culture. The patient was transfused with 300 cc. of citrated whole blood. The temperature rose to a normal level during the following 12 hours. The cyanosis disappeared 48 hours after the prontosil and prontylin were discontinued. (See Chart 1.)

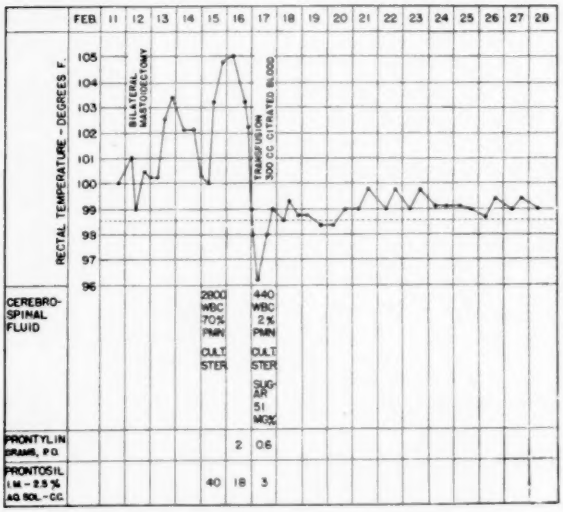

Chart 1. *Temperature chart and data on Case 4.*

The time of onset of the sterile meningitis cannot be determined accurately because this patient had headache and elevation of temperature for at least two days before admission to the hospital. However, from the clinical course it seems more likely that it occurred postoperatively. While it is possible that spontaneous drainage of the focus causing the aseptic meningitis (coincident with the administration of prontosil and prontylin) may have accounted for the striking improvement, the rapid fall in temperature and the clearing of the cerebrospinal fluid, we believe that the improvement may be explained also by the destruction of the organisms in the focus.

Case 5. A. M., a white male, aged 9 years, was admitted to the Children's Hospital, March 10, 1937, with the complaint of discharge from the left ear of 2 weeks' duration following an upper respiratory infection. The patient was a well-developed and nourished boy, co-operative and in no acute distress. There was a profuse, moderately thick discharge from the left ear canal; the drum was red and thickened and presented an inferior perforation. There was a small, firm, tender swelling which was thought to be an infected lymph node behind the pinna on the mastoid process. The mastoid tip was tender to pressure. The tonsils had been removed. Lymph nodes were palpable on each side of the neck in the anterior and posterior triangles. The eye grounds were normal. Neurologic examination was negative. Urinalysis on admission showed a large trace of albumin, acetone and from 1 to 2 leukocytes per high-power field in the uncentrifuged sediment. Examination of the blood showed 3,500,000 erythrocytes and 13,800 leukocytes per cu.mm., 70 per cent of the latter being polymorphonuclear cells. Beta hemolytic streptococci were cultured from the aural discharge. The Mantoux test, with 0.01 mg. of old tuberculin, was positive. X-rays of the mastoids showed clouding and some evidence of bone destruction on the left.

The temperature did not rise above 100°F. on the day of admission. The following morning it was 100°F. and at noon it was 101°F. At 3 p. m. the boy complained of pain in the left ear, became restless and appeared acutely ill, and the temperature was 105.8°F. He appeared drowsy but was oriented and showed no signs of meningitis. No chills were noted. A culture of the blood was taken, and in 16 hours eighteen colonies of beta hemolytic streptococci per cc. of blood had grown. At 5 p. m. the left mastoid was opened and was found to contain pus and granulation tissue. A marked degree of bone destruction was present. Over the knee of the lateral sinus a large perisinus abscess with marked edema and infiltration of the vein wall at this point was noted. The bone was removed over a wide area until a margin of normal vein was visible on each side of the abscess site. The sinus did not pulsate, and seemed to collapse readily under the palpating finger. It was not disturbed further. The mastoid wound was left open, and the patient was given 500 cc. of 10 per cent glucose intravenously. Para-aminobenzenesulfonamide and prontylin were started after the operation. The temperature rose to 106.4°F., 7 hours after the operation. The patient became totally disoriented and so violently active that despite hypnotics forcible restraint had to be used. The following day the temperature was 105°F. and the patient was still delirious. It was difficult to administer prontylin so only one dose was given. However, he received prontosil intramuscularly and para-aminobenzenesulfonamide subcutaneously. He was given fluids intravenously and by a nasal tube. The 2nd postoperative day found him still irrational and much weaker. It was still necessary to administer fluids by nasal tube. The lips and nail beds were deeply cyanotic. During the next 3 days the condition of the patient steadily improved. He began taking fluids by mouth, and the temperature returned to normal after 48 hours. Henceforth recovery of the patient was uneventful. The mastoid wound remained clean, and healed in gradually by granulation. Because of the severity of the illness and the necessity of daily mastoid dressings the patient remained in the hospital for 1 month. (See Chart 2.)

This case is prescnted to demonstrate the marked fall in temperature, rapid sterilization of the blood stream and general improvement in a child gravely

ill with acute mastoiditis, perisinus abscess and septicemia, after simple mastoidectomy with exposure of the lateral sinus and institution of therapy with prontosil, prontylin and para-aminobenzenesulfonamide. In spite of the advanced pathologic changes in the parietal wall of the vein it was not molested beyond gentle palpation. The Tobey-Ayer test under anesthesia, immediately before operation, had given a normal reaction on both sides. With this information at hand, and believing that at worst an infected mural thrombus and a localized phlebitis were present, we decided to leave

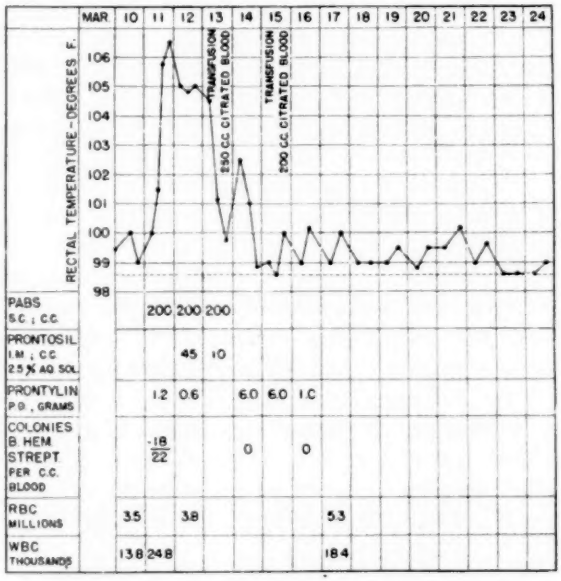

Chart 2. *Temperature chart and data on Case 5.*

the vein untouched and treat the patient expectantly.

Case 6. P. G., a white female, aged 5 years, was admitted to the Children's Hospital, April 22, 1937, with a complaint of a painful swelling behind the left ear, a discharge from the ear of 1 day's duration, and a fine, prickly red rash on the trunk of 3 days' duration. The temperature was 103°F. There was a dry, fading erythema on the anterior abdominal wall. A profuse thick discharge was noted in the left ear canal, and there was a red, tender swelling on the mastoid process. Diagnoses of scarlet fever, acute otitis media and a beginning subperiosteal abscess on the left were made. Beta hemolytic streptococci were cultured from the aural discharge. X-rays of the mastoids showed very slight haziness of the pneumatic structures on both sides, although the cells were not obscured and the intercellular trabeculae were distinct. Treatment consisted of hot-water compresses to the area behind the left ear and prontylin orally. The patient received an initial dose of 1.2 gm. of prontylin, followed by a maintenance dose of 0.6 gm. every 6 hours. During the next 3 days the temperature dropped to a level of from 99 to 99.8°F., the swelling over the mastoid process disappeared and the discharge from the left ear diminished. Beta hemolytic streptococci were cultured from the aural discharge on April 27 and 31. The temperature remained below 100°F. and the patient seemed to be recovering, but the ear continued to discharge purulent material. Cultures of the aural discharge were sterile on May 4 and 5. The temperature rose to 100.6°F. on May 7. The discharge from the left ear became more profuse, and on May 8 a swelling again appeared over the mastoid process. X-rays of the mastoids on May 10 showed diffuse clouding of the pneumatic structures, indistinct trabeculae on the left and normal structures on the right. A left mastoidectomy was done on May 11 and a subperiosteal abscess and a perforation in the cortex leading to the antrum were found. There was marked destruction of the cell walls, and the mastoid was filled with granulations and thick yellow pus. A portion of the lateral sinus covered with granulations was exposed, but the sinus was not thrombosed. Beta hemolytic streptococci were cultured from the mastoid. Following the operation the temperature rose to 101.8°F. and remained at this level for 24 hours, then became normal on the 2nd day following operation. The prontylin was omitted the day of operation but was started the day after. The patient was discharged to a hospital for contagious diseases on the 3rd day after operation.

This record is included in order that several important facts relative to the use of para-aminobenzenesulfonamide and its derivatives may be mentioned. This patient was treated with a dose of prontylin which was considered to be adequate so far as our present knowledge is concerned. Although the cultures of the discharge from the ear became sterile while the patient was receiving prontylin, a postaural abscess developed after two and a half weeks of treatment, and x-rays of the mastoids at this time showed marked destruction of the cells. Furthermore, at operation an opening in the cortex was found and widespread destruction of the cell walls was noted. The mastoid contained purulent material and granulations, and the beta hemolytic streptococcus was cultured from this purulent material. Bacteriologic study of the hemolytic streptococcus showed it to be an F variant,[6] which is one of the types observed by us to be susceptible to destruction by para-aminobenzenesulfonamide and its derivatives.

DOSAGE AND METHODS OF ADMINISTRATION

The plan of dosage of para-aminobenzenesulfonamide and its derivatives used at the present time at the Infants' and the Children's hospitals is as follows:

By mouth: Prontylin tablets; an initial dose of approximately 0.6 gm. per 20 lb. of body weight, followed by a maintenance dose of 0.06 gm. per lb. of body weight per 24 hours divided into four or six doses.

Intramuscularly: Prontosil solution (2.5 per cent aqueous); 1 cc. per lb. of body weight per 24 hours, divided into four or six doses.

Subcutaneously: Para-aminobenzenesulfonamide (0.8 per cent in physiologic salt solution); an initial injection of 100 cc. per 20 lb. of body weight,

followed by maintenance injections of 100 cc. per 40 lb. of body weight every eight to twelve hours.

Intrathecally: Para-aminobenzenesulfonamide (0.8 per cent in physiologic salt solution); 5 to 10 cc. less than the volume of cerebrospinal fluid withdrawn by lumbar puncture, injected once or twice daily according to the circumstances.

Prontylin tablets were given whenever oral administration was not contraindicated by nausea or vomiting. In such an event prontosil solution or para-aminobenzenesulfonamide was administered parenterally.

The concentration of para-aminobenzenesulfonamide in the blood of patients receiving the drugs under this schedule of dosage was found to be from 10 to 15 mg. per cent (Marshall's method[7]).

A rigid rule of dosage could not be adhered to in every case, as each patient presented an individual problem. In general, however, an attempt was made to follow the above method.

TOXIC REACTIONS

The toxic symptoms attributed to para-aminobenzenesulfonamide and its derivatives[1, 8, 9] are cyanosis, nausea and vomiting, fever, lassitude and headache, and hematuria. Unpublished reports have added hemolytic anemia, agranulocytosis, acidosis, skin rashes and jaundice.

It was our practice to decrease the dose or discontinue the drugs at the appearance of cyanosis in the first few patients treated with these chemical substances. With further experience, and after no ill effects could be demonstrated as a result of the cyanosis, the appearance of this symptom did not deter the use of para-aminobenzenesulfonamide or its derivatives.

Cyanosis of the lips and nail beds was observed in each patient of this group after receiving the drugs for from twenty-four to forty-eight hours. In each case the cyanosis disappeared twenty-four or forty-eight hours after the drug was discontinued.

In Case I, as may be seen in Table 1, there was a rather significant decrease in the erythrocytes of the blood after forty-eight hours of treatment. It is impossible to state definitely that the drugs produced this decrease, but the fact is suggestive. It is well known that rapid destruction of the erythrocytes may occur during the course of infections caused by beta hemolytic streptococci. It has been our plan in the treatment of such patients to use all possible therapeutic procedures, and we consider transfusions of citrated whole blood an aid in restoring the erythrocytes to a normal level.

Nausea and vomiting, fever, hematuria, agranulocytosis, acidosis, skin rash or jaundice was not observed in any patient of this group.

SUMMARY AND CONCLUSIONS

The protocols of 6 patients with severe complications, resulting from infection of the middle ear and mastoid with beta hemolytic streptococci, treated with para-aminobenzenesulfonamide and its derivatives, have been presented and commented upon. All patients recovered, and there was no evidence of sequelae.

Patients with middle-ear and mastoid infections who are being treated with para-aminobenzenesulfonamide or its derivatives should have careful, repeated physical examinations, and bacteriologic studies of the organism causing the infection. X-ray examinations of the mastoid at the onset and during the course of treatment should be used to determine the status of the mastoid infections.

It should be remembered that if these drugs are being used the dosage should be adequate, and they should be considered as an adjunct in the treatment of patients, not as the sole therapeutic agent.

From the many recent reports in the literature and from our observations of the use of the drug it is evident that we are dealing with a therapeutic agent the true value of which can only be determined through continued observations.

The prontosil, prontylin and para-aminobenzenesulfonamide used in the treatment of the patients at the Infants' and the Children's hospitals were donated by the Winthrop Chemical Company.

We express our thanks for permission to use the report of Case 3, which was treated at the Beth Israel Hospital.

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SURGICAL TRAINING IN THE NON-UNIVERSITY HOSPITAL

Presidential Address

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PROVIDENCE, RHODE ISLAND

A GENERAL hospital has for its duty the care of the sick in the community which it serves. This it accomplishes by the immediate care of the patients in its wards, by the training of nurses and doctors and by the advancement of medical knowledge. The old idea of the hospital as a medical boarding-house is fortunately long past. It is axiomatic that the more teaching there is in a hospital, the better is the immediate care of the patient. The hospital is supported by its community either directly by contributions, endowments and paying patients, or indirectly by taxation, as in the case of municipal hospitals. The community giving this support can in all fairness demand in return that the hospital shall so train doctors that the members of the community, both in and out of the hospital, will receive proper and expert medical care.

There are two phases to the training of the surgeon by the hospital: the intensive training of the man living in the hospital as intern or resident, and the long training when he is a member of the visiting staff. These are supplementary. For entirely aside from the fact that surgery is not static and each year brings its advancement, it is impossible to make a surgeon of a man by filling him with knowledge which may be poured out at will. Only the experience of years can give the ripened judgment which we call wisdom. We must be careful to look at the intensive training not as a short cut but rather as a firm foundation.

In the general hospital with a rotating service, all the interns receive some months of surgical training. This training is quite elementary, because of the short time available. The average intern is so busy with the number of new things he encounters and in acquiring technic—the "how things are done"—that there is little time for him to acquire the "why things are done." As a consequence, having done a few operations under a guidance not often realized, he graduates filled with that little dangerous knowledge which leads him to undertake cases beyond his powers, and so the community suffers. This is the sort of "trained" man the hospital now presents to the community. It is often no fault of the man; for, as I have said, there is no opportunity to further his immediate training. It is here that the average general hospital fails in its opportunity. The hospital by its present intern system does make possible the sorting out of those who should be encouraged and helped to advance in surgery. It is unfortunate that the tangible, dramatic and financial (the last too often illusive) aspects of surgery often make their appeal to those who are without aptitude of mind or fingers.

Given the selected man, how shall further intensive training be furnished him? It must be by some form of resident service. The details of this service must vary with the hospital. There are various successful types of long standing, which may furnish a pattern. The question of the number of beds necessary to furnish an adequate resident service is similar to that required in maintaining a training school for nurses. The number will vary with the size and location of the community to be served. The majority of hospital graduates settle near their hospital. Other details, which also must vary, such as the number of years of service, inclusion of special services and relation to staff, we shall not discuss.

There are certain principles in regard to residents which are pertinent. First, as to the necessity of the residents' being paid: it is curious that the career of medicine, the preparation for which is the most expensive in time and money, is about the only one that has an unpaid apprenticeship. To continue this unpaid apprenticeship for several years longer than is now required is unfair. To so lengthen the period of training that a man is about thirty before he can begin to earn a living may work a hardship, deprive the worthy of deserved opportunity, or throw this line of work open only to those who are financially independent. None of these are desirable things. This matter of expense is the great stumbling block for the hospital. The average hospital is without sufficient funds to meet all the demands upon it. There is the ever-present deficit. An endowment for residents similar to scholarships for students in the university is the desired thing.

There are several possibilities for making resident service a thoroughly postgraduate affair. It could be made a part of an established graduate school of medicine, the residents being enrolled in it and the school having supervision over their training. However, the choice of men for resi-

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dencies would have to remain with the hospital. Or, somewhat after the English system, each resident could still be enrolled as a student of the medical school from which he graduated. There would not be enough residents at any one time to make this a burden on the school. A higher degree might be conferred at the termination of this period of training.

An objection to the establishment of a resident service is that it makes the intern service less interesting and less desirable. It undoubtedly does so, but chiefly by taking away from the intern some things, such as operating, which he should not be doing. This deprivation should be more than compensated by the more continuous instruction that the intern would receive and that he really needs. That any one hospital might have difficulty in obtaining interns under such a system would be true unless other hospitals adopted a similar system.

The adjustment of the visiting staff would be troublesome at first. But perhaps, especially for the younger man who now does some of the work of a resident, the system would help to prove that supervision of work is of value and that operating is only a part of surgery. It is not enough to furnish opportunity to a resident, though to a large extent a man must train himself. There must also be guidance so that he will not waste time but will progress according to plan. This is the immediate duty of the visiting staff. Those not interested in teaching and developing the younger man should have no place on the hospital staff.

Let us now consider the opportunity that should be furnished by the hospital for the training of the surgeon on its visiting staff. This old and tried system has developed and will develop, without residencies, many good surgeons, as witness the membership of the New England Surgical Society. But I think, in looking back, that those of us who had no opportunity of being residents regret the lack of such a foundation.

The proper size of the visiting staff in proportion to the number of beds is a nice question. It varies from a small number of full-time members of a clinic to the large staff of the open hospital. Both have their place in the care of a given community. For the advancement of surgery and the training of teachers, a full-time specialized staff is essential. For the development of surgeons to care for the community, which is the function of the usual general hospital, the larger staff is necessary. A small staff with residents can care for a hospital service better than a large staff, but it cannot care for all the individuals who require surgery in a sizable community. The average patient prefers, and will go to, a surgeon who gives him personal attention before, during and after operation. There should be enough men receiving the training of staff work to cover the needs of the community. Of what use is the advancement of surgery if it cannot be applied? How often it is said that such and such a procedure should only be done by the most experienced surgeon. How can anyone become the most experienced surgeon unless as a younger man he is given opportunity? Experience is not inherited. To maintain a fairly large number of men in the junior staff positions is desirable. These positions should be trial ones, with advancement, as opportunity offers, to those who have been proved. And no staff position should be regarded as a fixture. The age limit for hospital service is a proper and desirable thing. For most positions it should be lower than at present.

Every hospital should furnish proper surgical instruction to those who will eventually be members of the visiting staff. For the time is coming when hospitals will not be on the approved list unless their visiting surgeons hold certificates from the American Board of Surgery.

76 Waterman Street.

IODINE RESPONSE AND OTHER FACTORS IN THEIR RELATION TO MORTALITY IN THYROTOXICOSIS

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BOSTON

IN STUDYING the records of patients with hyperthyroidism in order to determine the frequency of response to iodine in this disease, it became apparent that the mortality was in large measure related to the response or failure of response to iodine. A careful analysis was therefore made of the data, and it forms the subject matter of this paper.

The response to iodine in hyperthyroidism has been extensively studied in the Metabolism Laboratory and Thyroid Clinic of the Massachusetts General Hospital since Plummer first introduced the use of iodine in 1923. There were 1187 records available for analysis, dating from 1924 to July, 1935. The metabolic data of each individual were scrutinized to determine the change produced by iodine, and judgment was made, in most cases, from the levels of metabolism rather than from individual tests. A drop of 15 points in the higher brackets, and of 10 points in the lower brackets, was considered a positive iodine response. Other facts which might have a bearing on the iodine response or on mortality were recorded. These were correlated with respect to response to iodine. Finally, the records of those patients who died were analyzed in order to determine all the possible causes of death and their relation to therapy.

RESULTS

Iodine Response. Of the 1187 cases studied, 731 furnished sufficient data to determine the response to iodine. Of these, 706, or 96.6 per cent, showed the usual drop in basal metabolic rate, and 25, or 3.4 per cent, showed no significant change in metabolism, or at most a doubtful change. There remain 456 cases in which there were not enough data to show the effect of iodine. As indicated in Table 1,

TABLE 1. *The Frequency of the Response to Iodine in 1187 Patients with Hyperthyroidism; the Mortality Rate in Relation to Iodine Response.*

RESPONSE	CASES		MORTALITY	
	No.	*%*	*No.*	*%*
Positive iodine response	706	96.6	8	1.1
Negative iodine response*	25	3.4	6	24.0
Total	731		14	1.9
Unknown iodine response	456		13	2.9

*Includes negative and doubtful cases.

there were 8 deaths in the group that responded to iodine, a mortality rate of 1.1 per cent, and 6 deaths, or 24 per cent, in the group which failed to respond. The operative mortality rate was 1.3 per cent in the former group (8 deaths among 630 patients), and 24 per cent in the latter (6 deaths among 25 patients). There were 13 deaths in the unclassified group of unknown iodine response, 6 operative and 7 nonoperative. The patients operated on in the entire series totaled 782; there were 20 operative deaths, or a mortality rate of 2.6 per cent. It was impossible to determine the nonoperative mortality rate in this series because no attempt was made to follow any large number of such patients. The 7 nonoperative deaths occurred in very sick patients before operation could be attempted.

Sex and Age. The striking finding that almost half the deaths in cases with known iodine response occurred in the small group of patients (3.4 per cent) who failed to respond to iodine led us to analyze various factors which might be involved. Table 2 shows the distribution by sex of those pa-

TABLE 2. *The Distribution of Patients and Mortality Rate by Sex and According to Iodine Response.*

SEX	POSITIVE IODINE RESPONSE				NEGATIVE IODINE RESPONSE				ENTIRE SERIES			
	CASES		MORTALITY		CASES		MORTALITY		CASES	MORTALITY		
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*No.*	*%*	
Male	162	95.2	3	1.8	8	4.8	2	25.0	245	8	3.3	
Female	544	97.0	5	0.9	17	3.0	4	23.5	942	19	2.0	
Total	706		8	1.1	25		6	24.0	1187	27	2.3	

tients whose response is known. It is seen that relatively more males than females failed to respond. This difference is, however, statistically not significant. The mortality rate was also slightly different in males; it was twice as great as in females in the positive-response group, and nearly identical in the negative-response group. For the whole series, including patients with unknown response, the mortality rate was 3.3 per cent for males and 2.0 per cent for females. Similarly, the operative mortality rate for the entire series was 4.0 per cent of 175 male patients and 2.1 per cent of 607 female patients.

The distribution of patients by age groups, as shown in Figure 1, illustrates a higher incidence of negative response in the older age groups. For example, only 14.4 per cent of the positive group were fifty years or over, whereas 48 per cent of the negative group were in this age group. Expressed in another way, 10.5 per cent of the patients fifty years and over failed to respond, whereas only 2.1 per cent of those under fifty failed

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to do so. Figure 1 shows further that the mortality in both groups tends to increase with age. However, the relatively high mortality in the patients who did not respond to iodine persists in most of the age groups. Caution must be exercised in interpreting these figures because of the small number of patients in the negative group. Nevertheless, it can safely be stated that neither sex nor age accounts in any appreciable way for the difference

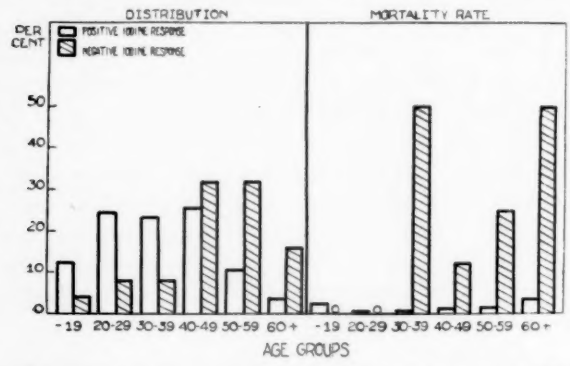

Figure 1. *The percentage distribution by age of 731 patients with toxic goiter with respect to their iodine response and the corresponding mortality rates. Of this number, 706 responded to iodine and 25 did not.*

in mortality between the responding and nonresponding cases.

Another factor analyzed was the initial basal metabolic rate level before iodine medication was started. Figure 2 reveals the interesting fact that

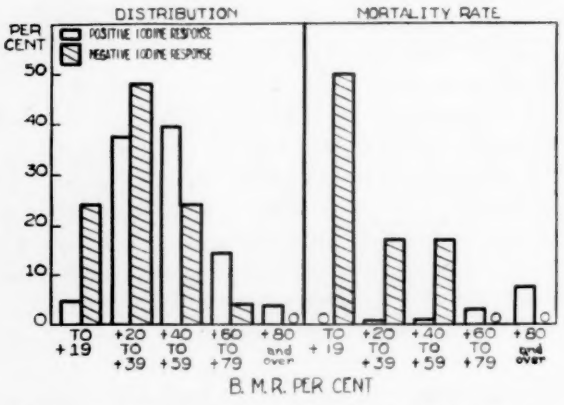

Figure 2. *The percentage distribution of 706 patients responding to iodine and 25 not responding, according to initial basal metabolic rates, and the corresponding mortality rates.*

the rate was lower in those who failed to respond than in those who responded. For example, 48 per cent of the negative group had metabolic rates below +30, as against 19.4 per cent of the positive group. In other words, the incidence of negative response was higher in the lower brackets of metabolism. The mortality in the positive group tended to increase with metabolism, as was to be expected, since the basal metabolic rate is usually a fair index of the severity of hyperthyroidism; on the other hand, in the negative group there were more deaths in those patients with metabolisms under +30 than in those with metabolisms over +30. In fact, there were no deaths in the 3 patients with metabolic rates above +50. It follows, therefore, that the metabolism in this group of patients is not a reliable index of the operative risk.

Type of Goiter. The thyroid glands of these patients were classified as either diffuse or nodular. As shown in Table 3, 60 of the patients who

TABLE 3. *Mortality in Relation to Character of Gland and Iodine Response.*

RESPONSE	TYPE OF GOITER	CASES No.	CASES %	MORTALITY No.	MORTALITY %
Positive iodine response	Diffuse	638	91.4	8	1.3
	Nodular	60	8.6	0	0
Negative iodine response	Diffuse	16	64.0	2	12.5
	Nodular	9	36.0	4	44.4
Entire Series	Diffuse	940	84.7	9	1.0
	Nodular	170	15.3	18	10.6

responded to iodine, or 8.6 per cent, had nodular goiters, whereas 36 per cent of the nonresponding group had them. In a few cases the character of the gland was not determined. There were twice as many deaths in the negative group with nodular goiters as in the same group with diffuse goiters, the rates being 44.4 per cent and 12.5 per cent respectively. The operative mortality rate for the entire series was 4.5 per cent in 111 patients with nodular goiter, and 2.2 per cent in 671 patients with diffuse goiter. The presence of a nodular goiter is not the fundamental explanation for this difference, because there were no deaths among those patients with nodular goiter who did respond to iodine. However, it is of importance to note that of the 9 patients with nodular goiter who did not respond to iodine, all were over fifty years of age, 7 had metabolism under +30 (the highest being +42) and two thirds had heart disease. All 4 deaths occurred in the latter group. Their metabolisms were +15, +18, +12 and +42 respectively.

Heart Disease. It is to be expected that heart disease would play an important role in mortality from hyperthyroidism, with or without operation. A few facts about the cardiovascular system which are measurable by clinical methods were selected for analysis. These are analyzed in Table 4. The data indicate that the type of iodine response was not influenced by the presence of cardiac enlargement. The frequency of cardiac enlargement was about the same in the patients that responded to iodine as in those who did not — 41 per cent and 48 per cent respectively. In both groups the mortality was greater in the patients with cardiac enlargement than in those without, but the differ-

ences were nearly proportional in each group. The actual values were 2.1 per cent and 41.7 per cent respectively for the positive and negative groups with cardiac enlargement, and 0.5 per cent and 7.7 per cent respectively for the positive and negative groups without it.

In contrast to cardiac enlargement, generalized arteriosclerosis, with or without demonstrable aortic involvement, occurred three times as often in patients who did not respond to iodine as in those who did. The mortality, also, was greater in both groups of patients with arteriosclerosis than in those without, but only proportionately.

The presence of cardiac arrhythmias, as a group, did not affect the response to iodine to any great extent. To be sure, the patients with preoperative auricular fibrillation (paroxysmal and constant) responded less often to iodine than those with normal rhythm. Four out of 61 patients with auricular fibrillation (6.5 per cent) failed to respond to iodine, whereas 19 out of 631 patients with normal rhythm (3.0 per cent) failed to do so. It is in these patients with auricular fibrillation that there was an appreciable increase in mortality as compared to those with normal rhythm. The increase, however, was nearly proportional in both positive and negative groups, so that the discrepancy in mortality in these two groups remained unchanged.

TABLE 4. *The Incidence of Cardiovascular Findings in Relation to Iodine Response and Corresponding Mortality Rates.*

Cardiovascular Findings	Positive Iodine Response			Negative Iodine Response		
	Cases		Mortality	Cases		Mortality
	No.	*%*	*%*	*No.*	*%*	*%*
Cardiac enlargement						
None	414	59.1	0.5	13	52.0	7.7
Generalized	208	29.7	1.9	7	28.0	33.3
Auricular	15	2.1	—	—	—	—
Ventricular	63	9.0	3.2	5	20.0	60.0
Arteriosclerosis						
None	580	83.0	0.9	11	44.0	9.1
Peripheral	63	9.1	1.6	8	32.0	12.5
Peripheral plus tortuous aorta	54	7.7	3.7	6	24.0	66.7
Arrhythmias						
None	612	87.4	1.0	19	76.0	21.1
Auricular fibrillation*	57	8.2	3.5	4	16.0	50.0
Paroxysmal tachycardia	5	0.7	—	—	—	—
Extra systoles	24	3.4	—	2	8.0	—
Heart block	1	0.1	—	—	—	—
Blood pressure (in mm.)						
Systolic						
-120	43	6.3	—	2	8.0	—
120-159	469	67.8	0.9	10	40.0	30.0
160-199	160	23.2	1.9	9	36.0	22.2
200+	19	2.7	5.3	4	16.0	25.0
Diastolic						
-60	69	10.0	1.5	2	8.0	—
60-79	359	52.0	1.1	9	36.0	22.2
80-99	241	34.9	0.4	10	40.0	10.0
100+	22	3.2	4.5	4	16.0	75.0

*Paroxysmal and constant.

The blood pressure, both systolic and diastolic, tended to be higher in the patients who did not respond to iodine than in those who did, but there was little correlation between the blood pressure and mortality in either group.

When one considers heart disease as a whole, with or without congestive failure, the results are interesting. As indicated in Table 5, the incidence of heart disease in the negative-response group was much higher than in the positive and unknown-response groups, the figures being 40.0 per cent for the negative, 9.3 per cent for the positive, and 9.7 per cent for the unknown. The frequency was unaffected by age, sex and type of gland. The mortality in each group was much higher in patients with heart disease than in those without, the results for the entire series being 14.2 per cent and 0.9 per cent respectively. As a matter of fact the greater portion of the high mortality in the negative group is accounted for by the deaths of those having heart disease, particularly of the 4 patients with nodular goiter previously referred to. Likewise, the operative mortality rate was greatly affected by heart disease, being 12.0 per cent for those with it and 1.3 per cent for those without. As for the 7 nonoperative deaths, 6 were directly due to heart disease, or at least heart disease played an important role in the final outcome. Undoubtedly the strain of long-standing thyrotoxicosis on hearts already damaged by disease produced cardiac failure.

Causes of Death. On searching for the causes of death of thyrotoxic patients, one is impressed by the dominance of two factors, namely the thyrotoxic "crisis," and cardiac failure. Both may be complicated by pulmonary infection. In any given case there are usually several factors operating to produce a fatality, with one factor dominant. In the patients who responded to iodine, thyrotoxic "crisis" was the predominant cause of death, being the underlying factor in 3 cases; cardiac failure was the cause in 2 cases and possibly in a third; 1 patient died of air in the pulmonary artery, and another of either tetany or bilateral adductor palsy. On the other hand, in the patients who did not respond to iodine heart disease alone was responsible for 2 deaths, and in 3 others was associated with evidence of partial "crisis." The sixth patient died of cellulitis and bronchopneumonia.

In the group as a whole, then, age and sex, the presence of nodular goiter, the coexistence of heart

TABLE 5. *Mortality in Relation to Iodine Response and Heart Disease.*

Condition	Positive Iodine Response				Negative Iodine Response				Unknown Iodine Response				Total			
	Cases		Mortality		Cases		Mortality		Cases		Mortality		Cases		Mortality	
	No.	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*	*No.*	*%*
With heart disease	66	9.3	3	4.5	10	40.0	5	50.0	44	9.7	9	20.5	120	10.1	17	14.2
Without heart disease	640	90.7	5	0.8	15	60.0	1	6.7	412	90.3	4	9.7	1067	89.9	10	0.9

disease, and most important, the response to iodine materially influenced the fatal outcome.

DISCUSSION

It is not intended to compare the mortalities from exophthalmic goiter in different clinics, nor to discuss the reason for their variation. The subject has been adequately presented by Means,[1] Boothby[2] and the Thompsons.[3] Attention is drawn to the fact that nearly half the deaths occurred in patients who did not respond to iodine, although this group constituted only a small portion (3.4 per cent) of all patients with thyrotoxicosis. As compared with the positive-response group, the negative one included more patients over fifty years of age; the average basal metabolic rate was lower; nodular goiter was more frequent; and the incidence of heart disease was higher. Although it was not possible to determine accurately the duration of the disease in each group, these traits of the negative group indicate that in it the disease was of longer duration. Particular attention is called to the nonresponding patients with heart disease, because they contributed a large portion of the deaths.

The data presented here focus attention upon the importance of iodine response. However, it is not to be inferred that this is the only factor to be taken into account in judging the operative risk or the ultimate prognosis. Numerous other elements influence prognosis, and some of these have been discussed. The state of nutrition and psychic disturbances are also extremely significant.

SUMMARY AND CONCLUSIONS

In a series of 1187 patients with toxic goiter, of whom 731 had adequate metabolic data, it was found that 3.4 per cent failed to respond to iodine. The mortality rate in this group was 24.0 per cent, whereas in the responding cases it was 1.1 per cent.

The immediate causes of death were many, and several factors usually operated in any given case. The most important factors were thyrotoxic "crisis," with or without pneumonia, and cardiac failure. The former was relatively important in the patients responding to iodine, and the latter in the nonresponding ones.

Several factors bearing on iodine response and mortality were analyzed, and a rough parallelism was noted. Males responded less frequently to iodine than females, and had almost twice their mortality. Both the failure to respond to iodine and the number of deaths tended to increase with age. Most of the negative responses fell in the lower brackets of metabolism, and likewise the deaths; consequently the metabolism could not serve as an index of operative risk in this group. On the other hand, in the responding cases the metabolism was a useful guide to operative risk because the mortality varied directly with the level of metabolism. Both nodular goiter and heart disease were more common in the nonresponding cases than in the responding. Similarly, mortality was higher in patients with nodular goiter than in those with diffuse goiter, and in patients with heart disease than in those without it.

None of these factors adequately explain the failure to respond to iodine and the high mortality in that group of patients. However, a large portion of this mortality occurred in a small group of patients characterized as being relatively old, running relatively low metabolisms, possessing nodular goiters and showing some form of heart disease.

It is hoped that by concentrating effort on this small group of patients who do not respond to iodine, the mortality in toxic goiter can be appreciably lowered.

370 Commonwealth Avenue.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23511

PRESENTATION OF CASE

A ten-year-old, American born, white schoolboy entered the hospital complaining of swelling of the legs of eight months' duration.

About two years before entry his parents noticed that he tired more easily and became blue and short of breath on exertion. He was unable to sleep flat in bed and developed nocturia of two or three times a night. His physician made a diagnosis of congenital heart disease and restricted his activities. One day a few months later he had a headache and vomited two mouthfuls of bright-red blood. He was put to bed for a few days. His condition remained essentially unchanged for the next year. Eight months before entry his legs began to swell gradually, and the orthopnea became more severe. He was put to bed for a week, and the swelling entirely disappeared. However, three months before entry swelling of the legs again appeared and persisted in spite of bed rest. The edema gradually spread upward, and by the time of entry had involved his back.

He had had chickenpox at three, whooping cough at five, and measles at eight years of age. He had never had nosebleeds, growing pains, swelling or pain in his joints, recurrent sore throats or any cardiorespiratory symptoms previous to his present illness. His physical and mental development had been normal. The family history was negative.

Physical examination revealed a well-developed but somewhat undersized, dyspneic boy, sitting up in bed. There was moderate cyanosis of the lips. The chest was barrel-shaped with bulging of the precordium, and the sternum and ribs were quite tender to percussion. The lungs showed fine crepitant rales over both bases, and diminished to absent breath sounds, dulness, and diminished tactile fremitus over the right base posteriorly. The expansion was poor, and the breathing labored. The neck veins were enlarged and pulsating. The pulse at the wrist was of poor quality, and the blood pressure could not be determined. The heart was enlarged to the left axillary line with the apex impulse felt well inside the area of dulness. The right cardiac border was percussed 5 cm. outside the midsternal line. The sounds were of fair quality, the rhythm was regular, and a presystolic gallop could be heard over the entire precordium, except at the apex. P_2 was louder than A_2 but not markedly accentuated. No thrills or murmurs could be made out. The abdomen was protuberant, and a definite fluid wave could be elicited. The liver edge was slightly tender and palpable three fingers below the costal margin. There was marked edema of the legs and sacral region.

The temperature was 99.5°F., the pulse 90. The respirations were 27.

The urine had a specific gravity of 1.030 and contained the slightest possible trace of albumin. The blood showed a red cell count of 4,650,000 with a hemoglobin of 75 per cent. The white cell count was 9150, 88 per cent polymorphonuclears. An electrocardiogram showed right axis deviation, inverted T_3, high P_1 and P_2, and depressed S-T_2.

He was treated with digalen, mercupurin and morphine but responded very slowly. He had some diuresis, but the edema spread to his penis and scrotum. On the fourth day a portable x-ray of the chest showed the heart to be grossly enlarged, particularly to the right. It was the shape of a water bottle, the margins were smooth and sharp, and the usual chamber outlines were not seen. The left main bronchus was slightly elevated, and the angle of the carina was increased. There was increased density at both bases obliterating the outline of the diaphragm. The exact position of the aorta was not established. In spite of the drugs the edema failed to clear, and he gradually became more dyspneic and cyanotic so that an oxygen tent was necessary. On the seventh day the presystolic gallop was replaced by a reduplicated second sound. He died the following day.

DIFFERENTIAL DIAGNOSIS

DR. EDWARD F. BLAND: This is indeed a puzzling case, and I am certain I cannot explain all the clinical features outlined in this history. On the other hand, it seems to me that we can pick out two essential features of the clinical course.

The first one consists of dyspnea and cyanosis on exertion appearing only recently in a boy of eight, who presumably had previously been well. These two manifestations persisted and ultimately progressed to severe heart failure, which we must assume was predominantly of the right side. It seems to me the two presenting manifestations of dyspnea and cyanosis may give us a clue and perhaps suggest that the difficulty was either in the heart or, possibly, in the lungs. In any child of ten years, dying in this part of the world with heart failure, one cannot dismiss, without serious consideration, rheumatic infection as the most likely cause, but it seems to me we can exclude that in this instance for a number of reasons. In the first place, it is very difficult to explain the initial

period of a year and four months of exertional dyspnea and cyanosis on the basis of rheumatic infection. Secondly, we have no evidence from the clinical course that there was an infection or, for that matter, even an inflammatory process. Thirdly, there is a complete absence of suggestive rheumatic symptoms; and, finally, in the group of 306 fatal cases of rheumatic fever in childhood, which Dr. T. D. Jones and I have recently studied, not one of them failed to have cardiac murmurs during the fatal illness. I am willing to discard rheumatic infection without further comment.

It seems to me the second possibility that demands careful consideration here is some obscure and silent congenital lesion of the heart. If this is the answer, it is certainly not one of the usual types with which we are clinically familiar. I have considered first the possibility of an "idiopathic" hypertrophy, either congenital or unknown, but the age of the patient and the late appearance of symptoms and signs are distinctly against it, especially the absence of symptoms for a period of eight years. It would be a little difficult to explain the predominantly right-sided heart failure on this basis alone, and furthermore, the patient died without any obvious superimposed infection. I am unable to exclude, however, a slight interatrial septal defect, but we have no proof of such, and if present, I do not believe it was an important contributing factor.

It seems to me a third factor that might possibly be to blame is pericardial disease of a subacute inflammatory nature. We have seen such occasionally in older people which extended over a period of several months, and when we had an opportunity to observe it at postmortem examination, there was marked thickening and chicken-fat appearance of the pericardium. There are a number of factors against such a possibility. In the first place, it would again be difficult to explain the year and four months' dyspnea and cyanosis on this basis. In the second place, we have no evidence of infection of an inflammatory process, and in spite of suggestive clues scattered through the record it seems to me that the size of the heart alone is convincing evidence against a chronic or subacute constricting process. And, further, the heart sounds are too good, and we have no supporting evidence from the electrocardiogram in the form of low voltage and T-wave changes. I cannot exclude, in view of the subsequent course, the fact that this child may have had a complicating hydropericardium since he had fluid in the other body cavities, but I do not believe that the pericardium is primarily to blame. Therefore, we have to look for disease elsewhere in the body.

It is obvious that we are not dealing primarily with a renal disorder. The urine had a normal specific gravity, there was only the slightest possible trace of albumin, and nothing more is said about the urinary picture. Furthermore, since he was treated with mercurial diuretics we must assume that the kidneys were not suspected. He had no anemia, and we have no reason to think that there was a profound nutritional disorder as a possible cause of heart failure.

So far as I am concerned, that leaves the lungs as the most likely seat of trouble. The child died presumably of cor pulmonale with failure. We know that the usual causes of chronic cor pulmonale are not present in childhood, namely chronic fibrosis of the lungs, emphysema of many years' standing, long-standing bronchitis or pneumoconiosis, or extensive pleural adhesions. It usually takes years for chronic cor pulmonale to develop after chronic pulmonary disease. Finally, I am left with only one alternative which we know does occur in childhood — an obliterating endarteritis of the pulmonary arterioles. It is a rare condition, and I therefore hesitate to suggest it, but it seems to me that it more nearly explains the entire course here, and especially the presence of cyanosis from beginning to end. Ayerza described the disease in 1901. He found in some of his cases that syphilis apparently was to blame. I personally have observed 2 cases. We know that the condition may occur in young people. It usually occurs, however, in young adults. The first case that I recall was a young girl who died in her early twenties in this hospital. We had studied her for a number of years and thought that she had a congenital lesion of the heart. The case was complicated by the presence of systolic and diastolic murmurs at the pulmonic area. She ultimately died of right-sided heart failure, and the postmortem showed that the entire difficulty was due to obliterative disease of the smaller pulmonary arteries. The second case simulates this one in some respects — a boy eleven years of age died abruptly in 1931. His symptoms of dyspnea and slight cyanosis on exertion did not appear until two years before death. He, also, was mistakenly diagnosed as having congenital heart disease — again on the basis of systolic and diastolic murmurs along the left of the upper sternum, in addition to his symptoms.

I hesitate again to suggest this diagnosis because of its rarity, but it seems to me that, in a case as obscure as this, if one is to make a positive diagnosis, I should prefer to say that this patient had chronic cor pulmonale with right-sided heart failure of extreme degree and dependent primarily on obliteration of the pulmonary arterioles.

A PHYSICIAN: Is the x-ray picture of any help?

DR. BLAND: I felt that we had to discount some of the findings because the child was apparently quite ill at the time of the observations here and died within a week, but I should like to have Dr. Hampton's opinion.

DR. AUBREY O. HAMPTON: I am afraid it is not going to be of much value in your differential. I cannot add much to what the note has said. Certainly the left main bronchus appears high. That would mean dilatation of the left auricle ordinarily, though I suppose the left bronchus might be elevated if the patient had pericardial effusion, or simply a huge heart. So we cannot use that as evidence that the left auricle is dilated since we have such a huge heart picture. The hilus shadows are obscured by the heart shadow; that is, they are behind the heart silhouette and all we see is the peripheral blood vessels in the upper lobe. The larger vessels are dilated.

DR. BLAND: Will you tell us about the pulmonary conus?

DR. HAMPTON: I cannot see it. Certainly the shape of the heart is typical of pericardial effusion. One could not get a better water-bottle shape than that. The trouble is we did not fluoroscope the patient. There is another difficulty in that the right auricle may dilate to this extent and so a patient with decompensation may appear to have pericardial effusion.

DR. BLAND: We see that shape in children with severe rheumatic disease also.

DR. WYMAN RICHARDSON: What about polycythemia?

DR. BLAND: When it is present, cyanosis is a prominent feature. This child had been sick for eight months with congestive symptoms, and as I recall, in the child of eleven who died in 1931, polycythemia was not a prominent feature. I am just taking a long chance in order to make a positive diagnosis.

DR. RALPH W. DAFFINEE: This case was about as baffling in life as it is on paper. The outstanding thing on clinical observation was that here was a boy with marked evidence of right cardiac failure with nothing to account for it in the history. We usually get a story of infection setting off most forms of decompensation in childhood. We ought to have some history of rheumatic infection if it were due to rheumatic heart disease. He made practically no response to all the drugs we gave him. He had a tremendous diuresis, but fluid reaccumulated faster than we could get rid of it. Our diagnosis was congenital heart disease, but we could not specify what the lesion was.

DR. BLAND: I intended to emphasize the fact that all therapy seemed to fail; this I took to be a little bit more evidence of pulmonary-artery disease because that is our usual experience with therapeutic measures when dealing with cor pulmonale.

DR. PAUL D. WHITE: In 1931 we were impressed by the lad, of whom Dr. Bland speaks, with the cor pulmonale and endarteritis obliterans of the lungs, and if we had seen this present case within a short time after that we probably would have deduced what Dr. Bland has just presented. In the meantime we have seen a number of patients with congenital auricular septal defects that resembled this. Dr. Bland was in Europe when the patient came in. Dr. Sprague and I saw this boy when we were giving our graduate course. Dr. Louis Gross was one of the class, and I think we all came to the conclusion that the diagnosis was probably congenital heart disease with an interauricular septal defect. We did not think so far as Dr. Bland has.

DR. HAROLD L. HIGGINS: I do not believe that the cyanosis was as bad as Dr. Bland seems to feel. Cyanosis usually appeared only on exercise, and when he was in here he was a little dyspneic but never markedly cyanotic.

DR. TRACY B. MALLORY: I think that was also true of the dyspnea; it was only exertional.

DR. WHITE: We also thought of a triloculate heart. We had once seen a child with a single ventricle, but that seemed less likely here because of the electrocardiographic evidence.

CLINICAL DIAGNOSES

Congenital heart disease — triloculate heart or auricular septal defect.
Bronchopneumonia.

DR. BLAND'S DIAGNOSES

Chronic cor pulmonale.
Right-sided heart failure (extreme degree).
Arteritis obliterans of the pulmonary arterioles (Ayerza's disease).

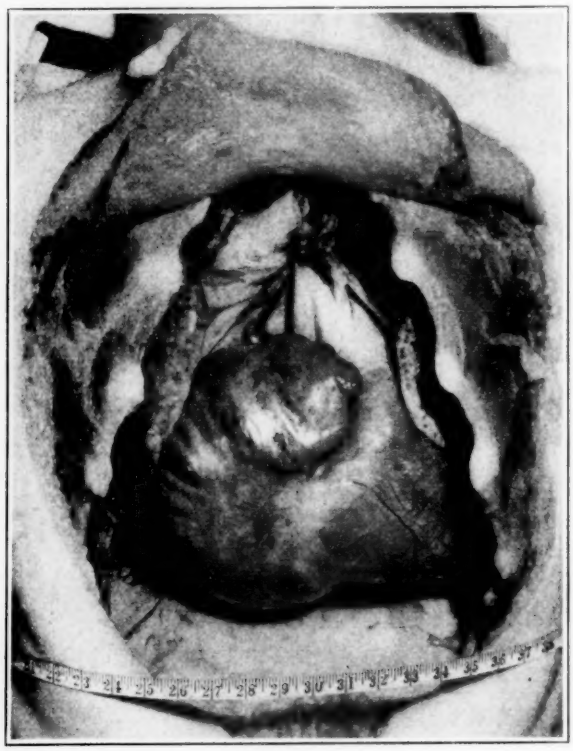
ANATOMIC DIAGNOSES

Cor pulmonale, etiology unknown.
Fibrosis of the endocardium of the left auricle.
Intimal fibrosis of pulmonary arterioles, venules and veins.
Interstitial edema of the lungs.
Infarct of the lung.
Chronic fibrous pneumonitis.

PATHOLOGICAL DISCUSSION

DR. MALLORY: It is fairly easy to describe what we found at autopsy but very much more difficult to interpret it from the point of view of its pathogenesis. The illustration — a photograph made after removal of the sternum and incision of the pericardium before the relations of the organs had been disturbed — shows clearly why the x-ray shadow had the so-called water-bottle shape. The

right auricle is greatly distended and makes up a much larger proportion of the presenting surface of the heart than is usual. The right ventricle also is large, but most of it is hidden behind the margin of the left lung. The entire heart has been rotated 30 or 40°, in a counter-clockwise direction, so that its greatest diameter is transverse rather than vertical. This rotation causes the pulmonary artery to run in a primarily vertical rather than horizontal direction, thus obliterating the usual prominence of the pulmonary conus. He had, of course, a very marked cor pulmonale. The right ventricle was markedly dilated and markedly hypertrophied. The left auricle was very much dilated as well as the right. The left ventricle was



Case 23511. *Cor pulmonale.*

the only normal chamber in the heart. The valves were entirely negative to minutest scrutiny.

A quite surprising finding when we opened the heart was the appearance of the left auricle. It showed an extreme grade of fibrous thickening of the intima with one or two spots of actual calcification. In sections through the left auricle the fibrous thickening of the intima was almost as thick as the muscle of the auricle itself. Marked intimal thickening of the left auricle is not rare in rheumatic mitral disease but it is usually irregular and patchy in character, whereas this seemed perfectly diffuse and uniform. Before I forget it let me add that none of the microscopic slides show the slightest evidence of rheumatic infection.

Another interesting finding was the very peculiar appearance of the lungs. They were very heavy, rather less crepitant than normal, and they felt a little tough and fibrous, though there was no consolidation except for a single small infarct. From the cut surface very little fluid exuded, rather to my surprise since their external appearance had led me to expect them to be wet.

On microscopic examination it was evident that there was a severe involvement of the smaller blood vessels throughout the lung. Anyone who has ever studied histologic sections of the lungs will realize how very difficult it is to identify as arterioles or venules the small blood vessels. The arterioles are almost identical with the venules in the extent and distribution of both the elastic fibers and the muscle cells. Dr. Brenner,[1] who worked on the subject several years ago in this laboratory and later wrote a monograph on it, practically gave up trying to distinguish them unless he could trace a connection with a larger artery or vein.

In this case there is an obliterative process going on in the majority of the smaller blood vessels, some of which I am sure are arterioles and more of which I am confident are veins and venules. The intimal thickening in some of the larger veins is quite marked and one is tempted to connect that up with the marked intimal thickening of the left auricle, because in many animals, the rat and mouse for instance, the pulmonary veins are functionally part of the left auricle, as shown by the extension of cardiac muscle fibers far out into the lung along all the pulmonary veins. I am inclined, therefore, to think that this fibrotic process which involves the small vessels in the lung is probably part and parcel of the one that caused the intimal thickening of the left auricle. The exact character of these vascular changes I shall postpone describing for a moment until one other set of findings has been covered.

The pulmonary parenchyma is by no means normal. The alveolar walls are thicker than normal, showing distinct edematous separation of their various elements and in many areas an increase in collagen. The alveolar sacs, however, are free from fluid as one would expect from the dryness of the cut surface. Desquamated alveolar cells are very numerous but few of them contain iron pigment, and this perhaps is the feature which best serves to distinguish the picture from the "cyanotic induration" of ordinary chronic passive congestion. The pleura and the interlobular septa are markedly edematous and the lymphatics are uniformly dilated. A few foci of true scarring — secondary to organization of either pneumonic patches or of infarcts I presume — are present but they do not seem numerous enough to have been of functional significance.

To sum up, then, we have a combination of cardiac, pulmonary vascular and pulmonary parenchymal changes. Which is cart and which is horse? Could everything be secondary to chronic pneumonitis? I think we can eliminate that possibility. Were the vascular changes primary? The right ventricular hypertrophy and the scars in the lungs would be reasonable sequelae, but the left auricular fibrosis is left unexplained. Could the auricular lesion have been primary? I believe it is worth a thought, though I know of no such recognized syndrome. The findings, however, fall well within the limits of that vaguely delimited syndrome known as Ayerza's disease.

The posthumous build-up of a widely touted syndrome about the single "black cardiac" whom Ayerza described in an unpublished lecture in Buenos Aires in 1901 is paralleled only by the case of Hanot and his liver cirrhosis. But the vagueness of the term is perhaps its justification since it allows one to group together all cases of cor pulmonale in which the etiology is not clear.

Dr. Bland has mentioned the 2 other cases which we have had in this hospital. I have had the opportunity of studying still another case referred to me from Iowa. The findings are different in all 4. The first case — the woman of twenty-six — had, I feel sure, congenital lues in spite of her negative Wassermann, since her teeth were typically Hutchinsonian and her liver showed the deep scars of a hepar lobatum. Her major pulmonary arteries showed extensive atheromatous deposits, but the small arteries and arterioles of her lungs were perfectly normal. The boy of eleven showed an active endarteritis of the medium-sized and small arteries, but his arterioles and alveolar walls were perfectly normal. The case from Iowa showed marked muscular hypertrophy and slight intimal thickening of all the arterioles but no changes in the larger vessels. Today's case shows fibrous intimal thickening without atheromatous deposits and without evidence of active inflammation of small arteries and arterioles and of venules and veins of all sizes. I might add that none of these lesions conform to the picture of pulmonary endarteritis in mitral stenosis so well described by Parker and Weiss.[2]

Surely, with such a variety of histologic pictures there can be no question of a single disease entity, of a single etiology. Let us continue to call them Ayerza's disease so long as we realize that in so doing we are confessing our ignorance.

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CASE 23512

Presentation of Case

First Admission. A forty-nine-year-old, white, native, assistant postmaster entered the hospital with the complaint of pain in the epigastrium.

About a year before entry the patient began having occasional attacks of indigestion, which he described as the raising of gas, accompanied by a cold shiver and followed by epigastric pain. The pain started as a dull ache in the epigastrium and radiated to the left, becoming sharper. Occasionally it radiated to the right, and sometimes it would start high up beneath the sternum and radiate down to the epigastrium. Often it went through to the back, and he could get some relief by applying a hot water bottle to his back. He found that he was more comfortable lying with his legs drawn up. He thought that the pain was more apt to come on when his stomach was empty. On several occasions the pain awakened him from sleep in the early hours of the morning. The pain was not definitely related to eating or relieved by food, but was somewhat relieved by soda. For a short period he obtained some relief by taking a milk diet. During the six months before entry his appetite was somewhat impaired. He had lost about 10 lb. in weight. There had been no change in bowel habits, and he had had no nausea or vomiting. On two occasions shortly before entry he thought he felt a lump in his epigastrium.

His past history and family history were essentially noncontributory.

Physical examination revealed a well-developed and nourished male appearing to be in good health but complaining of epigastric pain. The examination was essentially negative except for slight tenderness to deep pressure high in the epigastrium under the costal arch. The blood pressure was 130 systolic, 78 diastolic.

The temperature was 98.6°F., the pulse 80. The respirations were 20.

The urine examination was negative.

A gastrointestinal series showed a dish-like ulceration, measuring 7 cm. in diameter, slightly above the middle of the lesser curvature of the stomach, immediately below the cardiac orifice.

The patient refused operation and was discharged on the fourth day.

Second Admission. After discharge the patient became depressed at the prospect of operation and disappeared for about three weeks. He was finally located in New York and was brought back to Boston to re-enter the hospital. During the three weeks the symptoms previously described became considerably more severe. The substernal and epigastric pain made it almost impossible for him to eat. He finally agreed to have the operation.

The physical examination was essentially the

same as before except that there were a few fine rales at both lung bases.

The temperature was 98°F., the pulse 85. The respirations were 20.

A guaiac test on the stool was negative.

On the second hospital day an operation was performed.

Differential Diagnosis

Dr. Horace K. Sowles: The x-ray report confirms the fact that we are dealing with an ulcerating lesion in the stomach and localizes it on the lesser curvature, where both ulcer and carcinoma are most apt to occur. An ulcer that size of course suggests malignancy, although Dr. Hampton tells me that the x-ray department has seen many large ulcers in this area on the lesser curvature which were not malignant. Cancer would be more likely if the lesion were on the greater curvature or low down in the prepyloric area on the lesser curvature. This is one case in which a gastric analysis might have helped. The presence of free acid would have meant very little one way or the other, but its absence would have been strong evidence for cancer.

The substernal pain is of some interest. Of course it might be due to coincident heart disease, but I see no reason to believe so. If it is due to the same process as the epigastric pain, it suggests obstruction at the cardia. By x-ray the lesion does not extend that high, so I think that the pain may be passed off as an intermittent cardiospasm. On the basis of the character of the pain and the lack of anorexia, nausea and weight loss I am slightly in favor of ulcer as against cancer.

The patient's indigestion seems to have been a matter of gaseous eructation and pain without nausea. The pain was apparently chiefly epigastric, though we must consider the fact that it was sometimes substernal and that it sometimes radiated through to the back. What the significance of a "cold shiver" associated with epigastric pain may be, I do not know. When the pain was not in the midline it seems to have spread to the left rather than to the right. A pain that awakened him from sleep was almost certainly not functional. The statements concerning its relation to food are equivocal but the relief afforded for a time by a milk diet and also by soda suggests that it was an ulcerative gastric lesion of some sort; whether it was benign or malignant is the question.

In favor of a peptic ulcer is the early morning pain, relieved somewhat by soda and by the milk diet. The persistent character of the pain, its radiation through to the back, and the relief which the patient experienced from drawing up his legs, all suggest a rather deeply penetrating ulcer which may have burrowed through into retroperitoneal tissues.

X-Ray Interpretation

Dr. Aubrey O. Hampton: On first glance at the stomach you do not see much of anything. It is quite normal looking when it is filled and without pressure. In other words the ulcer does not project beyond the outline of the stomach. It is a dish-like shallow ulcer, 7 cm. in diameter. That is the description and the picture of a malignant ulceration. The margin of the ulceration is 1.5 cm. in thickness. There is no gradual change due to an inflammatory reaction around it. It ends very abruptly.

A Physician: In an inflammatory ulcer would you expect to see spasm on the opposite side?

Dr. Hampton: You would expect more spasm in the antrum and the defect would fade away at the margin, similar to a boil or an ant hill. There would be a gradual change.

Dr. Tracy B. Mallory: What about the depth of a peptic ulcer if it were that extensive?

Dr. Sowles: An ulcer this large would extend into the surrounding area.

Dr. Hampton: It would be deep and undermining. It would be more likely jug-shaped than dish-shaped.

Dr. Mallory: The question always comes up whether the clinician wishes to accept the radiologist's opinion. Do you agree with him, Dr. Sowles?

Dr. Sowles: I think that the character of the pain is more suggestive of ulcer and that the x-ray, as Dr. Hampton says, is more in favor of malignancy. I do not see how one can tell which it is.

Dr. Mallory: You consider it still unsettled?

Dr. Hampton: I mentioned to Dr. Sowles before the discussion that we had had some huge ulcerations on the lesser curvature that were not malignant, but I do not think we have had many like this that were not malignant.

Dr. William B. Breed: May I ask what the preoperative diagnosis was?

Dr. Mallory: Carcinoma.

A Physician: Did he have anemia?

Dr. Mallory: Not of any significant degree.

A Physician: Did they do a gastric analysis?

Dr. Mallory: No.

Dr. Sowles: They mentioned one test on the stool for blood. I do not suppose one test means anything, but would not carcinoma be more apt to bleed?

Dr. Mallory: No, I do not believe that would help one way or the other.

I am sorry Dr. Miller is not here to tell about the operative findings. I will read his note: "A 15 cm. median incision was made. High up on the lesser curvature there was a large, shallow ulcer,

5 by 6 cm. in diameter. There were a few lymph nodes, but no nodules. Nothing else was felt in this region. The lower three fourths of the stomach was then removed, turning in the pylorus. The lower end of the remaining part of the stomach was half closed, and then the left half was anastomosed to the jejunum which was brought up behind the colon."

PREOPERATIVE DIAGNOSIS

Carcinoma of the stomach.

DR. SOWLES'S DIAGNOSIS

Ulcer of the stomach.

ANATOMIC DIAGNOSIS

Reticulum-cell sarcoma of the stomach.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The stomach which came over to us showed a very large and extremely shallow ulceration. At the maximum point the depth of the ulceration was only 3 or 4 mm., which is rather unusual for such a large lesion whether it is benign or malignant.

Microscopic examination showed that it was what we now are calling reticulum-cell sarcoma. Five years ago we would have reported it as lymphoblastoma without second thought. In the interval we have not only learned to distinguish it histologically from lymphosarcoma and Hodgkin's disease but have discovered that it has a somewhat different prognosis from the other forms of what we used to lump together under some term such as malignant lymphoma. It is on the whole, I think, rather less sensitive to radiation, but when it occurs in a position where it can be resected rather strikingly long cures have been observed. Dr. Frederic Parker, Jr., has been particularly interested in primary reticulum-cell sarcoma of the bone, and has demonstrated a large number of cures running from fifteen to seventeen years. We have had thirteen tumors of the lymphoma series apparently primary in the stomach that Dr. Gall has recently been reviewing, and of these, three are of this reticulum-cell type. Two of the patients who were treated with a combination of surgery followed by radiation lived practically four years, and one is still alive so far as we know. We have had one case of lymphosarcoma which, following resection, has shown a still longer survival period. She has gone eight years and is still in excellent health. Among the cases treated by radiation the longest period of survival was only three years, so that I think the argument in favor of resection of these tumors is quite convincing. How would you feel about it, Dr. Holmes?

DR. GEORGE W. HOLMES: If I were to answer from a knowledge of the known character of the tumor and pay no attention to statistics, I should say that radiation is the method of choice in these cases. But I am familiar with the statistics and must admit that they strongly favor surgery, and I think that is what we should do — treat them as surgical cases. One reason perhaps why we do not see good results from radiation in this group is that we are comparing them with surgical results. Certainly the reticulum-cell type of tumor does do better with surgery than other lymphomas. If you compare the two groups when both are treated with irradiation this difference would not appear. It seems more a question of the spread of the tumor than treatment of the local lesion.

A PHYSICIAN: Do these metastasize or reappear as a new process?

DR. MALLORY: In a number of bone cases it has reappeared in another bone five or six years later, with no other evidence of involvement. To me that strongly suggests the development of a new tumor of the same type, not metastasis. The tumor may metastasize in ordinary fashion, however.

DR. SOWLES: Is this type of ulcer by x-ray suggestive of reticulum-cell tumor?

DR. HAMPTON: Dr. William C. Martin looked up all the cases here and those reported in the literature, and he mentioned two or three things which help in differentiation. This case shows one of the points that he mentioned, namely marked thickening of the mucosa of the remainder of the stomach. One other thing he mentioned is the occurrence of two lesions in the gastrointestinal tract, for instance, one in the stomach and one in the duodenum.

DR. MALLORY: I should think that one's best chance would be a study of the mucosal relief because the tumor often tends to infiltrate in the mucosal layer for some distance beyond the zone of ulceration.

DR. HAMPTON: In some of these cases the mucosa of the stomach showed convolutions such as one sees in the brain. I do not know whether they are submucosal changes.

DR. MALLORY: In this case the infiltration extends only a couple of millimeters beyond the ulcer in the mucosa, and outside that zone the mucosa looks normal — not even severe gastritis is present.

DR. JOHN TALBOTT: In regard to these thirteen cases that Dr. Gall has gone over, do you call them all reticulum-cell sarcomas?

DR. MALLORY: No, only three.

DR. TALBOTT: Is the incidence of reticulum-cell sarcoma going up?

DR. MALLORY: It is going up a great deal in this hospital because we are now recognizing it.

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CHRISTMAS 1937

The statement recently made by a leading Protestant churchman that Protestantism is reaching the end of a cycle need cause no great concern, despite a journalistic flair for inaccuracy that attributed to the speaker the prophecy that Protestantism is coming permanently to its end. As a matter of fact all cycles in human thought and affairs, as well as all natural phenomena, eventually come to their logical ends, giving way to new and eventually, we trust, better cycles.

Indeed, it is not only Protestantism that has arrived at this transitional period. All Christianity is there; the interracial and international relations of man to man are again in jeopardy; the outworn social and economic policies that have been maintained between groups and classes are at the crossroads; the soberer intellects in medicine are discovering new ways by which, if hopes can be converted into deeds, the beneficences of science will spread to all the strata of human kind, and our profession itself will be rescued from the political and economic dangers that threaten it in the changes that are inexorably taking place. The progress of the world is over a patchwork of crossroads, and every era of apparent security is in fact only a period of preparation for the ending of one and the beginning of another cycle. The world is always changing and sometimes, perhaps, its inhabitants will accustom themselves to the idea.

It is natural to be timid in the face of the unknown; it is human to cling to old and tried customs, however imperfect, rather than submit too readily to a future that has not been revealed, and yet, as Arthur answered Sir Bedivere from the barge:

> The old order changeth, yielding place to new,
> And God fulfills himself in many ways.

Christianity itself has not failed, although man himself may fail repeatedly. The spirit of right living is never lost, although we may lose sight of it as we follow false idols and strive toward spurious goals. Even though we may enter the dark period of strife and suspicion and jealousy, we shall emerge into a new era where there is again "Peace on earth, and among men, good will."

LEGAL MEDICINE

Although departments of legal medicine have existed for over a century in many Continental countries, this aspect of the profession has received but scant attention in the United States. Such departments, with their institutes, in foreign universities have educated undergraduates in the medical and law schools, have trained men electing to continue in the specialty, have served as consultants to the police physicians in the smaller cities and towns, have furnished unbiased expert testimony to the courts, and have assisted the police in the detection of perpetrators of criminal acts. Their contributions to society have been and still are inestimable, largely owing to the fact that their staffs have always consisted of well-trained men.

In this country there has been no call for institutions of this type. With few exceptions the various states have clung to the antiquated coroner's system; there has been no incentive for the young graduate for instruction in legal medicine; and the police in the majority of districts have been content to receive no intelligent medical assistance in the solving of crime.

Through the combined efforts of the Massachusetts Medical Society and the Massachusetts Bar Association a state-wide medical-examiner system was established in Massachusetts in 1877. Under the act, there are two full-time medical examiners on salary in Suffolk County and a varying number of county examiners in other counties on a fee basis, all appointed by the governor for a seven-year period. The arrangement worked out so well that it has been adopted in whole or in part by all the New England states except Vermont. A similar system was established by New York City in 1918 and by Essex County (Newark), New Jersey, in 1927. With both the latter, appointments are from civil service lists, with the result that properly qualified men hold permanent office.

A bill to abolish the coroner system and to establish the examiner system was introduced into the Michigan legislature last spring. The county examiners are to be under the supervision of a state medical examiner — possessed of "special training in pathology and the investigation of violent deaths." It is proposed to create and maintain a central medicolegal laboratory or institute. Unfortunately, the chief examiner is to be appointed by the governor for a term of four years.

Nation-wide recognition of the value of the expert in legal medicine is eventually bound to occur, just as it has in the field of public health, and with this arises the problem of providing properly trained physicians. Harvard University has evidently appreciated the need, for it has recently created a full-time professorship in legal medicine at the Harvard Medical School. No statements have been made concerning the plans for the department, but evidently more is planned than the lecture courses that have been given for the past five or six years, and undoubtedly the aim is the gradual development of a department and institute which will compare with or surpass those in Continental countries. The University is to be congratulated on its foresightedness. Let us hope that the states which eventually adopt the medical-examiner system will also be foresighted and will include some provision in their legislative acts so that well-trained experts rather than politically friendly nonentities will be appointed to office.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary*
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Boston

Case History No. 51. Premature Separation of the Placenta at Term

Mrs. L. T., a twenty-one-year-old white woman, who had been married five years, entered the hospital at term September 14, 1937, complaining of a vaginal discharge of bright-red blood. Two hours earlier she had felt moderate cramp-like pains in her lower abdomen recurring approximately every fifteen minutes. One hour later while lying in bed, she felt a brisk flow and saw that the bed was covered with blood. She arose and in walking around noted that the floor was being stained with blood.

She had been first seen March 25, 1937. Her last catamenia began December 16, 1936. Nothing in her family history was relevant to her pregnancy. Anterior poliomyelitis had occurred in childhood with no residual paralysis. Her tonsils had been removed. There had been no history of cough, dyspnea or limitation of exercise. Her catamenia had been normal and regular, with a twenty-eight-day cycle.

At her first visit it had been learned that following an apparently normal prenatal period she had delivered normally a premature infant on June 29, 1933. During the present pregnancy she had gained 12 lb. Her blood pressure had never risen above 110 systolic, 62 diastolic. Her urine had concentrated to as high a specific gravity as 1.025, never having gone lower than 1.017; on occasion a slightest possible trace of albumin and from 5 to 10 white blood cells per high-power field had

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

been found. There had been slight headaches during early pregnancy. She was urged to drink milk and to take tablets of ferrous sulfate, but had not complied. For a week before entry she had suffered a moderately severe cold with cough.

Physical examination showed a poorly nourished woman of average height. Her skin had a muddy pallor. Her tongue was coated; the mucous membranes were pale; the teeth were moderately carious; and the tonsillar fossae were clean and empty. Her pharynx was moderately injected. The breasts were normal in size, and her nipples protruded well. The heart showed no enlargement to percussion. Rhythm was regular; sounds were normal. The chest showed normal expansion. Her lungs were resonant throughout, and the breath sounds were normal. The abdomen showed a symmetrical mass extending to within 4 cm. of the xiphoid. There was no tenderness at the costovertebral angle, over the appendix or in any other part of the abdomen. The fetal heart rate was 80, irregular and best heard in the left lower quadrant on admission at 11 p. m. Her pelvic measurements were normal. Rectal examination was not done. The introitus showed a small, steady stream of bright-red blood. X-ray of the abdomen revealed an apparently normal fetus. At 11:45 p. m. the fetal heart sounds were no longer heard. Her blood showed a red cell count of 3,100,000, hemoglobin 60 per cent (Sahli). She was taken to the delivery room and preparation for delivery by the most appropriate means was made with a double setup. The donors being immediately available, preparation for blood transfusion was also made. Under gas-oxygen anesthesia the patient was examined vaginally. The cervix was found to be partially taken up with the os dilated two fingers, the vertex presenting and the membranes unruptured; it was thought that a small section of placenta was at one margin of the cervix. Bleeding was not increased by the examination. The pulse was 90; blood pressure 98 systolic, 78 diastolic.

On return to bed her contractions increased in duration and frequency, but relaxation seemed to diminish. There was little bleeding at this time. Beginning at 3 a. m., the uterus became very firm and rather tender. Her pulse rose to 120. The blood pressure remained the same. Repeated catheterizations yielded no urine. At 6 a. m. her red blood count was 2,700,000, hemoglobin 50 per cent (Sahli). She was transfused with 500 cc. of citrated blood and taken to the delivery room, where she was examined vaginally under gas-oxygen anesthesia. At this time a catheterized specimen of grossly bloody urine was obtained, which showed a specific gravity of 1.005, a very high trace of albumin, and from 50 to 100 clumps of white blood cells per high-power field in addition to the red blood cells. The membranes were unruptured, the cervix partially taken up, and the os dilated two fingers, and no placental tissue was felt. A blood clot lay high and loosely attached within the cervix. No bleeding ensued as it was withdrawn. The membranes were ruptured, and a very tight scultetus binder was applied.

For about five hours her condition remained the same. At 12:30 p. m. she complained of persistent cramps, but the uterus showed little evidence of definite contractions in its state of continuous firmness. At 1:35 p. m. the head suddenly appeared on the perineum. At 1:47 p. m. she was delivered normally of a 6-lb. stillborn female baby; the placenta followed immediately with a large amount of old retroplacental blood. There was a moderate amount of fresh blood; the fundus was firm. She was placed on shock blocks, and at 2:30 p. m. was transfused with 500 cc. of citrate blood. Her blood pressure was 100 systolic, 70 diastolic. At this time her face was distinctly edematous, as were her fingers and ankles. It was non-pitting in type. The same evening her temperature rose to 103.8°F., and her pulse to 120. The right kidney area was tender. A systolic murmur was heard, best in the pulmonic area. The blood nonprotein nitrogen was 60 mg. per cent. The fluid intake was temporarily limited to 1500 cc. per day. The edema disappeared in a few days and fluids were forced.

The temperature gradually fell over a period of two weeks to normal. Her right costovertebral angle was no longer tender at the end of a week. In ten days the cardiac murmur was barely audible, and her color had gradually improved. Treatment was largely supportive. An anemia diet was prescribed, and 18 gr. ferrous sulfate was given daily. Injections of liver extract were administered every other day. Daily doses of 15 gr. potassium citrate were given for three weeks. On September 24 another transfusion was given, owing to persistence of anemia. Dilute hydrochloric acid (1 dr. three times a day) was started on September 22. On September 30 the nonprotein nitrogen reached a normal figure of 32 mg. per cent. A urea-clearance test eighteen days postpartum showed 33.9 cc. of blood cleared of urea per minute. A few days later the urine concentration test (Castle method) showed a night concentration of 1.008 and a highest concentration of 1.012. The red blood-cell count and hemoglobin remained just above 2,000,000 and 37 per cent, respectively, until September 24 when, following transfusion, they rose to 2,500,000 and 50 per cent. On October 1 they were 3,160,000 and

62 per cent, and by October 12 reached 4,360,000 and 75 per cent. On only one occasion early in the course was the white blood-cell count above 10,000, when it reached a figure of 15,000. Intravenous pyelography, a retrograde pyelogram and cystoscopy yielded no evidence of urinary tract disease.

The patient was discharged in good condition at the end of one month to return to the Outpatient Department in two weeks for further blood study and urine examination.

Comment. This typical case of premature separation of the normally implanted placenta deserves some comment on treatment. When the patient entered the hospital with a history of having bled profusely at home and with a fetal heart rate of 80 per minute, no heroic effort was made to perform a cesarian section on a mother, who was a poor surgical risk, to obtain a baby whose condition was questionable. Since she was bleeding very little, an x-ray of the abdomen was obtained in order to verify whether the fetus was grossly normal in case its condition might improve and cesarian section then be considered. The fetal heart rapidly disappeared, and the patient seemed to be starting in good labor. Examination at this point led to a mistake in diagnosis, as a large blood clot within the cervix was thought to be a separated portion of a placenta previa. As the patient was in labor and not bleeding, no further manipulations were performed, although assuming this to be a placenta previa, it would probably have been better to rupture the membranes at this point to prevent the possibility of further hemorrhage. In the light of subsequent events it probably would also have hastened the expulsion of the fetus. Some five hours later, after the uterus had become more tonic and labor poorer, another vaginal examination revealed the absence of placenta previa; the membranes were ruptured and a tight scultetus binder applied in place of the more time-honored and more inquisitional Spanish windlass.

After all, what we are attempting to accomplish is firm pressure on the uterus to prevent retroplacental hemorrhage from filling it. The use of rupture of the membranes plus a tight abdominal binder has been satisfactorily used for some years at the Dublin Rotunda Hospital in place of the former method. It is simpler; no foreign body is introduced into the uterus, and the progress of labor can be readily observed. It should again be stated that this treatment is applicable in those cases of premature separation of the placenta where the mother is in poor condition or the fetus is nonviable, deformed or dead.

"GREEN LIGHTS TO HEALTH"

The list for the first three months of the 1938 program of the broadcasts "Green Lights to Health," sponsored by the Massachusetts Medical Society and the Massachusetts Department of Public Health and given through the courtesy of Station WAAB at 7:30 p. m. on the dates mentioned, is as follows:

January 5 — Care of the Hair. Dr. C. Guy Lane.
12 — Scarlet Fever. Dr. Conrad Wesselhoeft.
19 — First Aid. Dr. Charles C. Lund.
26 — Child Behavior. Dr. Randolph K. Byers.

February 2 — Milk. Dr. Richard S. Eustis.
9 — Heart Disease. Dr. William H. Robey.
16 — Psychology of Chronic Illness. Dr. A. Warren Stearns.
23 — Headache. Dr. T. J. C. von Storch.

March 2 — Tuberculosis. Dr. John B. Hawes, 2d.
9 — Care of the Skin. Dr. Everard L. Oliver.
16 — Medical Research. Dr. S. Burt Wolbach.
23 — State Health Department Activities in 1937. Dr. Henry D. Chadwick.
30 — Physical Therapy. Dr. Franklin P. Lowry.

MISCELLANY

CRIME AND THE COMMUNITY FUND — ANOTHER REASON FOR SUPPORT

In Charlestown State Prison there are more than 300 convicts who began their criminal careers before the age of seventeen.

At the Sherborn Reformatory for Women 80 per cent of the inmates are serving time for offenses that could have been prevented or treated by private social service, according to Dr. Miriam Van Waters, the superintendent.

Six out of every ten of the 1000 delinquent boys studied by the Harvard Crime Survey had shown serious behavior difficulties before the age of ten. Nine out of ten of them came from homes almost wholly unfit for children. Three out of four had never belonged to supervised clubs or settlements or attended vocational classes.

The average cost for convicting one man of a felony is around $1500. That sum in a Community Fund pledge will bring the advantages of character-building and recreational organizations to 500 boys.

The cost of keeping a youngster in the right sort of foster home does not vary greatly from that of keeping a man in prison. But, as between a jail and a good home, as between boyhood and manhood, where lies the greater hope of reformation? And what comparison is there between the losses inflicted upon society by the hardened criminal before he lands in a state prison and by the

youngster before he is dealt with by discerning social service and provided with wholesome recreation, environment and character-building activities?

It is easier — and cheaper — to prevent crime than to suppress it.

Where lies the greater hope for such prevention, if not in constructive work with children and youth? Where lies the greatest hope for lessening the levies on the taxpayer assessed by these evils?

The protection, care and guidance of children; the building-up of wholesome life for youngsters and youth; the constructive, character-forming activities of settlements, clubs and youth agencies — all these are in the day's work of Community Federation agencies.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1937

Diseases	November 1937	November 1936	Five-Year Average*
Anterior Poliomyelitis	6	1	15
Chickenpox	972	949	1007
Diphtheria	9	23	74
Dog Bite	600	565	484
German Measles	39	45	59
Gonorrhea	589	488	528
Lobar Pneumonia	269	291	285
Measles	282	414	481
Meningococcus Meningitis	5	8	6
Mumps	266	358	364
Paratyphoid A	0	0	0
Paratyphoid B	5	1	0
Scarlet Fever	593	493	716
Syphilis	558	469	404
Tuberculosis, Pulmonary	226	272	281
Tuberculosis, Other Forms	24	27	30
Typhoid Fever	17	6	8
Undulant Fever	2	5	3
Whooping Cough	471	1095	743

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from Arlington, 1; Deerfield, 1; Fairhaven, 1; Norton, 1; Provincetown, 1; Worcester, 1; total, 6.

Diphtheria was reported from Chelsea, 1; Fall River, 3; Middleboro, 1; New Bedford, 1; Revere, 2; Watertown, 1; Worcester, 1; total, 10.

Dysentery, bacillary, was reported from Cambridge, 4; Medford, 1; Milford, 1; Wellesley, 26; Worcester, 5; total, 37.

Encephalitis, lethargica, was reported from Clinton, 1; Haverhill, 1; New Bedford, 1; total, 3.

Meningococcus meningitis was reported from Cambridge, 2; Chicopee, 1; Fitchburg, 1; Worcester, 1; total, 5.

Paratyphoid B was reported from Easthampton, 1; East Longmeadow, 1; Lowell, 1; Shelburne Falls, 1; Sunderland, 1; total, 5.

Septic sore throat was reported from Boston, 5; Cambridge, 1; Lynn, 1; Medford, 1; total, 8.

Trachoma was reported from Boston, 2.

Trichinosis was reported from Boston, 1; East Weymouth, 1; total, 2.

Typhoid fever was reported from Attleboro, 1; Boston, 2; Brockton, 3; Chelsea, 1; Chicopee, 1; Grafton, 1; Holyoke, 1; Newbury, 1; New Bedford, 1; Northampton, 1; Oak Bluffs, 1; Pittsfield, 1; Quincy, 1; Stoughton, 1; total, 17.

Undulant fever was reported from Orange, 1; Stow, 1; total, 2.

Diphtheria continued to show record low incidence for the fourteenth consecutive month.

The reported incidence of lobar pneumonia, whooping cough and scarlet fever was below the five-year average.

Pulmonary tuberculosis was reported at a record low figure for the second consecutive month.

The incidence of typhoid fever was above the five-year average.

The incidences of anterior poliomyelitis and meningococcus meningitis were not remarkable.

Tuberculosis (other forms) showed record low incidence.

Measles, mumps, German measles and chickenpox were reported below the five-year average.

The incidence of undulant fever was not remarkable.

Paratyphoid fever after a diminution in October resumed its record high incidence.

The reported incidence of animal rabies showed a decrease since October. There is still an unusually wide distribution of animal rabies for this year. New foci were reported in Acton, Dracut and Malden. Previously reported foci in Lowell, Templeton and Brookline were active.

CONNECTICUT NEWS

Trailer Sanitation Regulations

Sanitary problems connected with the use of trailers have received consideration by the State Department of Health with the result that on September 16, 1937, the Public Health Council adopted the following regulations as part of the Connecticut State Sanitary Code:

Regulation 126. The Sanitation of Trailer Coaches. The term "trailer coach" is defined as any of the various types of vehicles with motor power or designed to be towed with an automobile and adapted to human habitation either for the purpose of sleeping or eating or preparation of meals or both or designed or adapted to use as an office or for the purpose of carrying on business.

(a) All toilets in trailer coaches shall be provided with fly-tight receptacles for containing excrement. Toilet vents shall be screened. Trailer coaches equipped with flush toilets shall be provided with suitable underneath holding tanks of adequate capacity for storage of trailer discharges between emptyings.

(b) No liquid wastes, garbage, refuse matter or other waste material from any trailer coach shall be deposited on or within the limits of public highways.

(c) No trailer coach shall be parked on land within 250 feet of and draining toward any source of public drinking-water supply.

(d) Cleansing of receptacles for wastes and excreta from trailer coaches by dipping or rinsing in the water of any lake, pond or stream is prohibited.

(e) No liquid wastes or excreta from any trailer coach shall be disposed of other than by emptying into a public or camp sewerage system, a septic or chemical tank system or a cesspool, provided that in isolated localities remote from camps or habitations such wastes may be disposed of by burying in the soil with an earth covering of not less than six inches. No wastes shall be thus disposed of at a point less than 250 feet of and draining toward any source of public drinking-water supply, nor within 50 feet from the banks of any lake, pond, stream or water course not a source of public drinking supply, nor within 50 feet from any highway gutter.

On the same date, Regulation 109 of the State Sanitary Code pertaining to camp-ground sanitation was amended by the addition of Section H with regard to disposal of wastes from trailers at camps.

Regulation 109.

(h) In every camp where space for trailer coaches is rented or held out for rent or on which free occu-

pancy or camping of trailers is permitted to trailer owners or users, sanitary facilities approved by the local health officer shall be provided for the disposal of wastes from trailer sinks and toilets.

Hartford Hospital Plans Modern Building

Plans as now completed call for a new modern building for the Hartford Hospital to cost between $2,500,000 and $3,000,000, as soon as funds are available. This will include all new equipment and a $400,000 power house.

Culminating an intensive study instituted about a year and a half ago when the hospital became acutely aware of its limited facilities for handling an ever-increasing influx of patients, announcement of the project stated in part that "necessity for providing in this manner for greater capacity for giving to our medical and surgical staff and to our nursing department enlarged and improved facilities and for more economical operation cannot be overstressed." Forty two per cent of the buildings on the grounds have been in continuous operation for from 50 to 77 years. The new structure is planned not only because present ones are at a far-advanced age and need to be replaced, but also because a modern hospital is so different from those of other years that under the existing conditions it is virtually impossible to run the present institution efficiently according to modern methods. The Hartford Hospital has frequently been overcrowded, and consequently, almost every department has been hampered in efficiency. Since 1921 the number of patients has doubled. During the last fiscal year ending September 30 the hospital cared for 17,402 patients. The maximum number of admissions for any one day was 92; 2200 babies were born in the hospital during this period.

Hartford Hospital Staff Changes

After many years of service Drs. Alfred M. Rowley, Edward R. Lampson and Orin R. Witter have resigned from the active staff. These vacancies have been filled by Drs. Clinton D. Deming, Donald B. Wells and John A. Wentworth.

New appointments to the staff are as follows: assistant physician, John C. Martin, M.D.; assistant pathologist, Perry T. Hough, M.D.; clinical assistant pediatrist, Albert U. Peacock, M.D.; clinical assistant cardiologist, Maurice B. Thompson, M.D.; clinical assistant physicians-at-large, Francis L. Lundborg, M.D., Donald R. Hazen, M.D. and William F. Storms, M.D.; clinical assistant obstetrician and gynecologist-at-large, Loftus L. Walton, M.D.

Connecticut Public Health Association

The Connecticut Public Health Association met in Hartford December 1 for an all-day session. "Public Health Nursing," "Safeguarding the Food Supply," "The Crippled Children's Program," "The State Cancer Control Program" and a "Symposium on Treatment and Prevention of Pneumonia" were among the papers read and discussed. Considerable discussion was provoked by the presentation of the cancer control program by Dr. Charles L. Larkin, of Waterbury, chairman of the Tumor Committee of the Connecticut State Medical Society. Dr. Larkin advocated that special research facilities be established at Yale University School of Medicine with a twofold purpose in view; that is, that of training the physicians to become more expert in the diagnosis and treatment of cancer, and that of carrying on further experimental studies necessary for progress in the study of malignancy. This seems entirely possible since this medical school has recently been the recipient of a four-million-dollar fund for cancer investigation.

At the pneumonia symposium Dr. Millard Knowlton, director of the Bureau of Preventable Diseases of the State Health Department, urged the launching of a pneumonia program with a legislative appropriation of $50,000 that would care for the costs of five types of serums as well as increased laboratory expenses.

Deaths

VOLKENHEIM — Mendel Volkenheim, M.D., dermatologist, died suddenly in his office in New Britain on Saturday, November 6. He was in his fifty-fourth year. Born in Russia, Dr. Volkenheim was brought to this country as an infant and he spent his early years in New Haven. He attended Yale University and the Yale University School of Medicine where he was graduated in 1908. About twenty years of his medical practice was spent in New Britain, but for an interim of several years he practiced in Hartford. He retained his membership in the Beth Israel and Emanuel Synagogue in Hartford. Dr. Volkenheim had a general practice until about a decade ago when he became a specialist in skin ailments. He was consulting dermatologist on the staff of New Britain General Hospital and a member of the local, county and state medical societies, and of the American Medical Association.

He leaves two sisters, Mrs. Rose Malkinson, of Hartford, and Mrs. H. W. Evans, of New Haven; and two brothers, Louis M. Volkenheim, of New Haven, and Samuel Volkenheim, of Portland, Maine.

ROBBINS — Walter J. Robbins, M.D., a general practitioner for thirty-seven years, died at his home in New Britain on November 22 after a long illness. He was in his sixty-third year. Born in Philadelphia, he spent his early life there, graduating from the University of Pennsylvania with a degree of doctor of medicine when he was twenty-three years old. He served his internship at Grace Hospital in New Haven, practiced medicine in that city for two years and went to New Britain in 1900. He was a member of various medical societies and a Mason. He leaves his wife, Mrs. Maude C. Marshall Robbins.

KAUFMAN — Charles E. Kaufman, M.D., town health officer of West Haven since 1935, died suddenly at his home on December 3. He was in his fifty-eighth year. He graduated from Yale University School of Medicine in 1920. His widow and three sons survive him.

STAUB — George E. Staub, M.D., of New Milford, practicing in that town for more than forty years, son of the late Nicholas Staub, a former state comptroller, died November 24 in the Stamford Hospital. He was in his sixty-ninth year.

Dr. Staub was graduated from Long Island College Hospital in 1893 and began practicing medicine in New Milford two years later. He leaves two brothers, V. P. Staub, president of the New Milford Water Co., and Dr. Howard Staub, of Stamford; and three children, Nicholas, Burr and Elizabeth.

NOTES

The following teaching and research appointments at Harvard Medical School were recently announced: Arnold P. Meiklejohn, of Harpenden, England, research fellow

in medicine; Arnold DeF. Barton, of West Roxbury, assistant in roentgenology; Ross Vroom, Jr., of Hingham, assistant in roentgenology; David Rothschild, of Montreal, assistant in psychiatry; Hsi Wei, of Nanchang, China, research fellow in pediatrics; Chuan Fan, of Soochoo, China, research fellow in pediatrics; James E. Robertson, of Santa Monica, California, research fellow in medicine and assistant physician to the Huntington Memorial Hospital; Ronald H. R. Belsey, of London, England, research fellow in surgery; Edward L. Tuohy, of Chatfield, Minnesota, research fellow in pediatrics and child hygiene; John A. Reboul, of Marseilles, France, research fellow in physiology; Edward Hamlin, Jr., of Boston, assistant in surgery; George E. H. Schwiegk, of Berlin, Germany, research fellow in pharmacology; Ralph W. Hunter, of Hanover, New Hampshire, assistant in neurology.

The Waltham Hospital recently celebrated its fiftieth anniversary. Among those taking part in the exercises were two physicians, Dr. Alfred Worcester and Dr. Henry A. Wood, who have been closely connected with the institution since its founding in 1887.

CORRESPONDENCE

ADEQUATE MEDICAL CARE

To the Editor: It is no longer possible to ignore the fact that there is a problem in many communities of this country concerning the lack of proper medical care for the indigent; the availability of adequate medical care by those who can pay but who are unable, or inadequately able, to obtain it; and in some instances, the catastrophic effect of financial demands upon individuals in unusual cases of prolonged illness or hospitalization. We cannot afford to continue to take a negative stand in the solution of this problem. We have frequently stated what we do not want, and what we will not tolerate, but constructive criticism has been lacking. Some even stubbornly refuse to listen to discussion of any change.

Question number one seems to be, Is there everywhere in this country adequate medical care available to all the people at a price they can afford to pay? The findings of the American Foundation would indicate that the answer is No. It is true that these findings are not entirely statistical but are based on the observations and opinions of a varied group of medical men, but for the sake of not giving the lie to so great a number of excellent medical men, let us grant that the answer is correct.

When we admit that point, then we must immediately ask ourselves another question, Should anything be done about it? Answering that in the affirmative, the next questions are, What? and By whom? We do not want compulsory health insurance; we do not want a political bureaucracy to administer any system which concerns the medical care of the sick. We do not want any system that will curb the initiative of the individual physician — rather one that will preserve the competitive spirit and the desire to excel in order to survive; we do not want any interference with the freedom of choice on the part of the individual patient as to the physician whom he will have serve him; and we do not want any interference with the right to freedom of contract whereby the physician may choose whom he will serve.

The time will come when we must propose a satisfactory solution, or have an unsatisfactory one thrust upon us. If the latter, it will come from the higher units of political organization with their bureaucratic personnel, their inefficiency and waste, and their interference with the rights which as just stated should not be tolerated.

The problem should be handled independently, but in concert, by the smallest of the political units, or expressed in another way, by people and doctors of the smallest of the organized divisions of society. In other words, by the welfare departments of individual towns, or by county units in rural sections not incorporated.

I suggest that whatever legislation may be proposed shall be permissive and not mandatory. There are many units of society which would never have to use the right.

Let legislation be something like this:

Each town, or county where it is the only incorporated unit, may use a sum of money equal to 5 per cent of its annual tax total for the protection and maintenance of the health of its citizens.

These funds are to be administered by the already existing welfare units so no new bureau need be created.

These funds are to be used to furnish medical and, in special cases, nursing care to the needy, together with medical supplies and appliances. In cases where hospitalization is essential, the necessary hospital (ward) expenses may be paid. Immunization by individual physicians may be paid for.

In the event of an unusually prolonged illness in the families of those able to pay only for ordinary medical and hospital care, a part of the medical expense may be paid from this fund.

The patient shall have the right to select the physician to treat him, and preserving the essential right of contract, the physician may accept or decline such service at his discretion.

In the event that a community is too poor to care for its indigent and needy with this percentage of its annual tax, the amount in excess of this fund spent in such care shall be a legitimate charge against the state, and the welfare board making such necessary expenditures shall be reimbursed by the state.

Any part of this fund unexpended shall be carried as a reserve fund, and when it shall amount to a sum equal to the 5 per cent permitted by law, that amount may be withdrawn and used for that year, no appropriation being necessary in lieu thereof.

These essentials could be couched in legal terms acceptable to the lawmakers of the states and enacted into law.

The roots of the problem are best understood by those nearest the source. No exalted group of legislators would be inclined to draft such legislation if it originated with them. They would want all the trimmings and folderol of political control, and bureaus with their attendant expense for individual aggrandizement.

The group at the heads of our medical societies will be accused of the same lack of competence and understanding, unless they give an attentive and sympathetic ear to the expressions and desires of the rank and file.

Thus will be avoided state or social medicine, as it is generally understood;

Thus will the medical need of every community in this country be served;

Thus will the independence of the practitioner be maintained;

Thus will be preserved the essential self-respect of the needy requiring such aid;

Thus will be avoided the creation of a political bureau with its wastefulness involving the expenditure of funds needed for a specific purpose, and the responsibility will be left to the states where it belongs; and

Thus will be served the needs of a nation, and no entering wedge of federal control of the practice of medicine will be driven into the structure of medical practice.

With every cent spent as it should be and at the place it should be, is not this something for us to think about?

JOHN R. FOWLER, LL.B., M.D.,
General Practitioner.

125 Main Street,
Spencer, Massachusetts.

FIRST MEDICAL MOTION PICTURE FILM IN GREAT BRITAIN

To the Editor: In the *Observer,* an English Sunday newspaper, June 13, 1937, there was a short article on "Medical Films." The following week Dr. Stephen Rowland, 7 Langham-Place, Northampton, wrote to the same newspaper as follows: "One of the earliest, if not actually the first, medical film shown in Great Britain was brought from America in 1906 by Dr. Chase of Boston." According to Dr. Morgan B. Hodskins of the Monson State Hospital, these motion pictures were taken by Dr. Chase at the hospital in 1904 or 1905. An early French camera was used. Later the hospital employed a man from Springfield, who took several pictures of convulsions. This old film cannot be located, although at one time it was known to be in the possession of the Department of Diseases of the Nervous System of the Harvard Medical School. Although the film cannot be found and possibly, even if discovered, could not be shown on the modern projector, perhaps some reader of this note can identify Dr. Chase and explain how he happened to take the film to England, where it apparently created quite an impression.

HENRY R. VIETS, M.D.

6 Commonwealth Avenue,
Boston.

RECENT DEATH

PITTA — JOHN C. PITTA, M.D., of 57 Allen Street, New Bedford, died December 11. He was in his seventy-eighth year.

Early in his career, Dr. Pitta was decorated by the King of Portugal for smallpox research. He was appointed Portuguese vice-consul in New Bedford in 1901, a post he held for ten years. He married Anna Mackay DeAlmeida, whose father later became Viscount De Valle De Costa, Portuguese consul at Boston. Dr. Pitta was the founder of a tuberculosis camp, the precursor of the New Bedford Antituberculosis Association and the Sassaquin Sanatorium. A founder of the Portuguese Auxiliary Society, he was also a life member of the National Geographic Society, a member of the American National Red Cross, the Loyal Order of Moose, the Monte Pio Society and the New Bedford Medical Association. He was a fellow of the Massachusetts Medical Society and of the American Medical Association.

His widow, a son, Dr. Carl A. Pitta, and three daughters survive him.

REPORT OF MEETING

HARVARD MEDICAL SOCIETY

The October meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on the evening of October 26. Dr. Stanley Cobb presided. In accordance with the usual custom, two cases were presented.

The first was a twenty-six-year-old, single female, who had complained of convulsions for the past eleven years. These attacks began at the age of fifteen and usually commenced with a stiffening of the right arm which rapidly spread into a generalized convulsion accompanied by incontinence and preceded by a slight aura of extreme apprehension. During the past two years she had usually fallen during the attacks. Over the course of the present illness she had taken phenobarbital. She entered the Peter Bent Brigham Hospital in July, 1937, at which time the phenobarbital was withdrawn, and following this a severe series of convulsions set in. Skull plates were negative. Physical and neurologic examinations were normal. The total protein of the spinal fluid was 27 mg. per cent. The usual laboratory tests were normal.

The case was discussed by Dr. Cutler and presented as a typical example of idiopathic epilepsy. Dr. Sosman felt that the encephalogram showed slight cerebral atrophy. Dr. Cobb pointed out some of the interesting features of this case, particularly the facts that emotion was a frequent precipitating factor and that in this case, as in most cases of epilepsy, it was rare to have attacks during exercise. For this reason, Dr. Cobb felt that these patients should be kept active and occupied. He also emphasized the danger of sudden withdrawal of phenobarbital in such cases, since this procedure frequently leads to severe status epilepticus.

The second patient was a forty-nine-year-old, single woman, who complained of attacks of falling in the past year. At the age of five she had had poliomyelitis and diphtheria. Four years before entry, after the death of her father, she had become increasingly lethargic, and in the past four years she had had several attacks in which she became unconscious and fell. There was no aura or convulsions. Her basal metabolic rate was —16, and she was put on small doses of thyroid extract without effect. Physical examination showed her to be obese and to have a slight degree of hirsutism. The deep reflexes were exaggerated, and there was a sustained clonus, but no Babinski. Her blood cholesterol was 358 mg. per cent. Skull plates were normal, and an encephalogram showed advanced cortical atrophy. This case was discussed by Dr. Cobb and others; it was considered to be a case of recent encephalitis.

Dr. Frederic A. Gibbs was the first speaker of the evening. He started the discussion of "Epilepsy in the Light of Recent Brain-Wave Studies." He reviewed the history of brain waves and pointed out that the study of such waves had been particularly active since the pioneer work of Hans Berger in 1925, but that the beginning of this study dates back to 1870. Many slides were shown to demonstrate the type of charts made by recording the electric activity of the cortex under normal and abnormal conditions. The normal tracing shows a series of waves of various frequencies, most of which are in the range of ten to thirty per second. The voltage varies from 0.002 to 0.050 millivolts. The rhythmic activity of which these electrical waves are evidence is a characteristic of all central nervous tissue. The respiratory center shows such rhythmic activity. In general, the same agents which alter respiratory rhythms alter cortical rhythms in the same direction. In sleep, all cortical frequencies are slowed just as respiratory activity is slowed. In deep sleep, the cortex shows large slow waves which are the cortical equivalent of snoring in the respiratory center. These slow waves in the cortex are entirely independent of respiratory movements and, so far as is known, of activity in the respiratory center. In general, it may be said that when the brain waves are slow in frequency the body musculature is relatively inactive, and when the brain waves are fast the body musculature is active. The body is a recorder of brain potentials. A series of very rapid waves is seen in the tonic phase of an epileptic attack; while in the petit-

mal type of attack, there is an alternation of fast and slow waves. In normal people, the brain waves slow down when the eyes are closed; in an epileptic, the brain waves may slow down much more than is normal when the eyes are closed. The mechanisms which regulate the frequency of brain waves are defective in epileptics. The petit-mal type of attack gives a tracing very similar to that seen in a tracing made of the respirations during Cheyne-Stokes breathing, that is, an alternation of fast and slow components. In the third type of epileptic manifestation, the so-called "psychomotor type," the waves are slow. Infants and deteriorated epileptics show very slow waves. Slow waves are also seen in very deep sleep, in stupor and under anesthesia.

Dr. William G. Lennox continued the discussion of this subject and pointed out the ways in which the observation of the brain waves may be of benefit in studying epileptics. In the first place, these investigations have thrown new light on the physiologic pathology of epilepsy and really call for a new terminology. Dr. Lennox suggested the use of "paroxysmal cerebral dysrhythmia" instead of epilepsy. Brain waves recorded during a seizure permit a definite diagnosis of epilepsy. Secondly, the type of dysrhythmia is different for different kinds of seizures. Thirdly, patients with this disturbance may show the characteristic dysrhythmia even between seizures. Fourthly, a study of the waves may permit prediction of a grand-mal seizure many hours before its occurrence. Fifthly, the point of origin in the cortex can frequently be determined, and in certain cases this permits the surgeon to remove the offending portion. Sixthly, the effects of certain drugs and chemical states can be evaluated by studying the effect on the brain waves. Dr. Lennox showed tracings to demonstrate the quieting effects of carbon dioxide and of glucose.

It was generally considered that use of this technic is a definite step forward in the study of epilepsy.

Dr. Hallowell Davis discussed the paper and pointed out that, while in the study of the function of most of our other organs we have several means at our disposal, in the study of cerebral function there is only this one. He also stressed the prevalent change that occurs in sleep, in which there is a marked slowing of all the rhythms. Dr. Schwab spoke briefly on the importance of eliminating extra waves and pointed out that there appears to be no relation between the brain rhythm and the rhythm of the tremor in Parkinson's syndrome.

NOTICES

REMOVAL

STUART N. GARDNER, M.D., announces the removal of his office to 24 Chestnut Street, Salem.

CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

CLINIC	DATE	ORTHOPEDIC CONSULTANT
Salem	January 3	Harold C. Bean
Haverhill	January 5	Arthur T. Legg
Lowell	January 7	Albert H. Brewster
Gardner	January 11	Mark H. Rogers
Brockton	January 13	George W. Van Gorder
Pittsfield	January 17	Francis A. Slowick
Springfield	January 19	Garry deN. Hough, Jr.
Worcester	January 21	John W. O'Meara
Hyannis	January 25	Paul L. Norton
Fall River	January 31	Eugene A. McCarthy

BOSTON DISPENSARY

A luncheon meeting of the clinical staff of the Boston Dispensary will be held in the auditorium on the second floor of the Dispensary building, on Tuesday, December 28, at 12 o'clock noon.

At 12:30 p. m., a symposium, under the auspices of the Cancer Clinic, will be conducted on "Cancer of the Breast."

OLIVER G. TINKHAM, M.D., *President,*
ALLEN R. BARROW, M.D., *Secretary.*

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The ninety-seventh semiannual meeting of the Essex North District Medical Society will be held at the Community Cafeteria, 131 Merrimack Street, Haverhill, on January 5, at 6:30 p. m.

PROGRAM

Business meeting at 7:30 p. m.
Principles and Proposals. Dr. Channing Frothingham.
Discussion.

Members of the Massachusetts Medical Society are invited to take part in the discussion.

W. D. WALKER, M.D., *President,*
E. S. BAGNALL, M.D., *Secretary.*

SPECIAL MEETING OF NORFOLK DISTRICT MEDICAL SOCIETY

A special business meeting of the society will be held in the Amphitheater of the Beth Israel Hospital, Boston, on Tuesday evening, December 28, at 8:15 p. m. Telephone Bea. 4400.

The order of business will be as follows:

1. Reading of the notes of the last meeting.
2. Report of a committee appointed by the President on order of the Massachusetts Medical Society for the purpose of investigating and reporting to the society regarding minimum fees throughout the district with particular reference to the fees now being received for services to welfare and old-age-assistance cases.
3. To hear speakers who approve and disapprove of "Principles and Proposals," as advocated by a group of some 430 physicians and surgeons of the United States. To discuss the subject and propound questions concerning it after the regular speakers have been heard.

It is hoped that every member of this society who is in any way connected with or interested in the medical care of welfare and similar cases will come to this meeting or will acquaint Dr. Walter A. Lane, chairman of the committee, with his views relative to the continued care of such cases at fees reduced from the minimum adopted by this society.

It is also hoped that those interested in "Principles and Proposals" will attend, present their points of view, and ask such questions as they may desire. Those not familiar with this subject will find the copy printed on pages 798-799-800 of the November 11 issue of the *New England Journal of Medicine*. Editorials relative to this subject will be found in subsequent issues of this *Journal* and in the various issues of the *Journal of the American Medical Association*.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, December 27

Tuesday, December 28

12 m. Boston Dispensary, clinical staff luncheon meeting. Auditorium.

8:15 p. m. Norfolk District Medical Society. Beth Israel Hospital.

Wednesday, December 29

*12 m. Clinicopathological conference. Children's Hospital Amphitheater.

*Open to the medical profession.

December 28 — Boston Dispensary clinical staff luncheon meeting. Page 1060.

January 4 — Greater Boston Medical Society, Beth Israel Hospital, 8:30 p. m.

January 9 - March 27 — Harvard University Free Public Lectures. Page 1012, issue of December 16.

January 13 — Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, 8:30 p. m.

February 2 — Second National Social Hygiene Day. Page 1013, issue of December 16.

February 14 — American Board of Internal Medicine. Page 969, issue of December 9.

April 4-8 — The American College of Physicians. Page 41, issue of July 1.

May 31, June 1 and 2 — Annual meeting of the Massachusetts Medical Society. Hotel Bradford, Boston.

October 17-21 — Clinical Congress of the American College of Surgeons, New York City.

District Medical Societies

BRISTOL SOUTH

May 5 — 5 p. m., New Bedford.

ESSEX NORTH

January 5 — Page 1060.

ESSEX SOUTH

January 5 — Page 1015, issue of December 16.

February 2 — Council Meeting, Boston.

February 9 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John B. Hawes, 2d. Subject: Dust and Disease.

March 2 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

April 6 — Gloucester Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

May 5 — Censors meet at Salem Hospital, 3:30 p. m.

May 11 — Annual meeting, Salem Country Club, Peabody. Dinner at 7 p. m. Speaker and subject to be announced.

FRANKLIN

Meetings will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesdays of January, March and May.

HAMPDEN

Meetings will be held on the fourth Tuesday in January, April and July.

MIDDLESEX EAST

Meetings will be held at the Bear Hill Golf Club, Stoneham, at 12:15 p. m. on January 12, March 16, and May 11.

MIDDLESEX NORTH

Meetings will be held at the Vesper Country Club, Lowell, on January 26 and April 27.

NORFOLK DISTRICT

December 28 — Special meeting. Page 1060.

January 25 — Hotel Kenmore. 8:15 p. m. The Management of Uterine Prolapse. Dr. Louis E. Phaneuf.

February 23 — Hotel Kenmore. 8:15 p. m. Dermatitis Venenata Due to Cosmetics and Industrial Irritants. Dr. John G. Downing. Discussion by Dr. Francis P. McCarthy.

March 29 — Hotel Kenmore. 8:15 p. m. Subject to be announced but to be related to diseases of the kidney. Dr. Albert A. Hornor.

May — Annual meeting.

The censors meet on the first Thursdays of May and November in each year.

NORFOLK SOUTH

Meetings held at 12 noon.

January 6 — Norfolk County Hospital, South Braintree.

February 3 — Norfolk County Hospital, South Braintree.

March 3 — Norfolk County Hospital, South Braintree.

April 7 — At the Quincy City Hospital.

May 5 — Annual Meeting.

PLYMOUTH

Meetings will be held at 11 a. m. on January 20, March 17, April 21, May 19 and July 21.

SUFFOLK

January 19 — Joint meeting with Boston Medical Library.

March 15 — Joint meeting with Boston Obstetrical Society.

WORCESTER

At the following meetings, except the annual meeting, dinner will be at 6:15, to be followed by business session and scientific program.

January 12 — Worcester City Hospital, Worcester.

February 9 — Worcester State Hospital, Worcester.

March 9 — Memorial Hospital, Worcester.

April 13 — Hahnemann Hospital, Worcester.

May 11 — Afternoon and evening, annual meeting. Place and schedule of program to be announced.

BOOK REVIEWS

International Clinics. Edited by Louis Hamman. Volume 2. Forty-seventh series. 315 pp. Philadelphia, Montreal, London: J. B. Lippincott Company, 1937. $3.00.

This, the second volume in the forty-seventh series of *International Clinics,* continues the excellent tradition of previous volumes and presents medical subjects of great practical interest without sacrifice to thoroughness or intellectual appeal. Every one of the articles will be of distinct interest to the general practitioner or internist who is still equipped with an inquiring mind. The thoroughgoing nature of such articles as those on "The Co-Existence of Syphilis of the Aorta and Bacterial Endocarditis," by F. J. Smith, "Actinomycotic Infection of the Central Nervous System," by E. D. Friedman and H. H. Levy, "Chronic Appendicitis," by B. J. McLoskey, and "Hemolytic Jaundice," by J. C. Sharpe, is refreshing when one considers the careless and cursory nature of the material which is frequently published under the headings of "Clinics" and "Contributions." Many practical facts of great value to the general practitioner are also presented. Thus one reads of the "Eye in Medical Diagnosis"; "Atypical Forms of Amebic Dysentery"; "The Care of the Breast and Nipples in Pregnancy and the Puerperium"; "Indications for Splenectomy in Medical Practice"; and "The Management of Asymptomatic Heart Disease." The volume is completed by full reports of recent progress in the treatment of puerperal infection and in the surgery of the biliary tract. The illustrations are unusually good and the index is adequate. The reviewer recommends this book without reservation to those who want full value in postgraduate medical education.

Emotional Adjustment in Marriage. Le Mon Clark. 261 pp. St. Louis: The C. V. Mosby Company, 1937. $3.00.

In recent years there has been a steady output of books dealing with love and marriage. All these have necessarily covered much the same ground, but their method of presenting the problems involved has varied widely. Dr. Le Mon Clark's book seems to us one of the best. The author writes well; his approach is dignified yet clear; the most sensitive reader can peruse the book without shrinking. There is a philosophic basis to his presentation of the subject which keeps before the reader's mind the relation of sex to the evolution of man and the development of civilization.

The greater part of the book is concerned with the sex instinct, its aberrations and its proper method of gratifi-

cation. The author believes that control of conception is essential to a successful marriage; he discusses this phase of the sexual life in considerable detail. His observations on marriage and divorce are sound, although his point of view as to the importance — or rather unimportance — of sexual fidelity is broader than the one that is generally held. This book may be recommended, especially for the more intelligent among prospective brides and grooms.

Chronic Rheumatic Diseases: Being the third annual report of the British Committee on Chronic Rheumatic Diseases appointed by The Royal College of Physicians. Edited by C. W. Buckley. 313 pp. New York: The Macmillan Company, 1937. $3.50.

This third volume of reports consists of a collection of original articles and reviews on the clinical aspects, etiology, pathology and treatment of chronic arthritis. The articles, as is pointed out in the foreword by Professor F. R. Fraser, reflect the activities of the committee appointed by the Royal College of Physicians. The volume serves to clarify what is known, what is conjectured and along what lines further research in the field of rheumatic diseases should be directed. An article by C. C. Okell will be helpful to those who have no adequate familiarity with the indications and limitations of vaccine therapy. The rationale of sympathectomy as a symptomatic measure in selected cases is discussed by J. R. Learmonth. J. Race, in an article on vitamins and rheumatic diseases, points out that in various chronic rheumatic diseases, especially in rheumatoid arthritis, the amounts of carotinoid pigments and vitamin A in the serum are subnormal, and a similar condition obtains in regard to vitamin C. A deficiency of vitamin B is also probable, but has not yet been demonstrated. There are reviews of recent contributions on osteoarthritis and other joint diseases, on diet and on chemotherapeutic measures. The volume is useful in making available to the profession at large the results of recent investigations.

Quelques Vérités Premières (Ou Soi-Disant Telles) en Chirurgie Abdominale. H. Mondor. 96 pp. Paris: Masson et Cie, 1937. 24 Fr. fr.

This small volume on abdominal surgery is one of a series of seven already compiled and three more in press. Acknowledging that certain notions which are accepted today as truths will in the future be shown to be fallacious, it is the hope of the editors that this work by Mondor presents statements regarding therapeutics and operations which are sufficiently proved to be permanently accepted. The editors' intention is to direct the student in the evaluation of symptoms and other clinical data and to list for the practitioner certain rules which he should not lightly disregard.

The type is italics and the arrangement in paragraphs of varying lengths suggests a series of aphorisms. The paragraphs are grouped under three major captions: "Abdominal Wall, Stomach, Duodenum, Pancreas"; "Intestine, Peritoneum"; "Gynecology."

Each paragraph is designed to teach a lesson — *a truth*. Under the various subdivisions of the major captions the paragraphs carrying these lessons are arranged more or less according to a general plan. There may be, for example, one or more paragraphs or lessons on gross pathology, on symptoms, on signs, on the necessity or urgency for surgery, or other special features which a particular lesion may indicate.

An insight into the character of the work may be suggested by noting the nature of some of the paragraphs. For example, under abdominal contusions are sixteen paragraphs teaching such lessons as: the severity of the case depends upon the presence or absence of visceral injury; death may ensue from shock or massive hemorrhage; peritonitis or hemorrhage may be of slow development; the early symptoms are not reliable in indicating the extent of the visceral damage; the apparent intensity of the trauma is not a certain measure of the number and gravity of visceral lesions; spasm of the abdominal muscles is more significant than some symptoms; morphine may mask signs; the abdominal wall is not always board-like; there may be varying degrees of muscular spasm; subcapsular and other hemorrhages may evolve slowly; and mortality mounts rapidly with delay.

Other paragraphs teach that vomiting may be incessant or absent, that fecal vomiting is a late symptom in acute intestinal obstruction, and that a metrorrhagia occurring after the menopause demands curettement and biopsy.

This volume is well printed on good paper and consists of 96 pages bound in cardboard covers. It contains much valuable advice and information. Its terse exposition and workable one-page index make it a good handbook for the student and general practitioner.

Crippled Children: Their treatment and orthopedic nursing. Earl D. McBride, in collaboration with Winifred R. Sink. Second edition. 379 pp. St. Louis: The C. V. Mosby Company, 1937. $3.50.

This book has been written primarily as a textbook of orthopedic surgery for nurses. The first ten chapters of the book on the nursing care of patients suffering from orthopedic disabilities and on the application of plaster and braces, are written in a clear, simple manner with a number of well-chosen illustrations. The remainder of the book is devoted to descriptions of the commoner diseases treated by the orthopedic surgeon. In the writing of textbooks for nurses, there is a tendency to fall into one of two errors, either the book is too concise and a clear picture of the disease is not given or it becomes too large and complex for the needs and understanding of the pupil nurse. The authors have at times fallen into the first of these errors. On the whole, the arrangement of the book is good. As a supplementary book to practical teaching in the nursing of the cripple it should be of distinct value.

Manual of the Diseases of the Eye: For students and general practitioners. Charles H. May. 498 pp. Fifteenth edition, revised. Baltimore: William Wood & Company, 1937. $4.00.

The present edition of May's Manual has been carefully revised and brought up to date.

The chapters on the ophthalmoscope and the ocular manifestations of general diseases have been rewritten, and other changes have been made in the text. There are several new illustrations.

This book is a very excellent textbook for the undergraduate student of medicine and for the general practitioner, and the reviewer recommends it highly.